"A STUDY ON P00 REVALANCE OF HELICOBACTER PYLORI INFECTION IN GASTRODUODENAL PERFORATION"

A Prospective study

DISSERTATION SUBMITTED FOR
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(GENERAL SURGERY)
Branch – I

MADRAS MEDICAL COLLEGE

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THE TAMILNADU DR M.G.R MEDICAL UNIVERSITY

APRIL-2014

CERTIFICATE

This is to certify that the dissertation titled **"THE PROSPECTIVE STUDY ON PREVALENCE OF HELICOBACTER PYLORI IN GASTRODUODENAL PERFORATION"** is the original work done by **Dr. M.ABDUL MALIQ**, postgraduate in the Department of General Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 to be submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai-32 towards the partial fulfillment of the requirement for the award of M.S. Degree in General Surgery, April 2013.

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This is to certify that this dissertation entitled **"A STUDY ON REVALANCE OF HELICOBACTER PYLORI INFECTION IN GASTRODUODENALPERFORATION IN RAJIV GANDHI DOVERNMENT GENERAL** is a bonafide and genuine research work done by **Dr. M.ABDUL MALIQ,** postgraduate student in the Department of General Surgery, Madras Medical College & Rajiv Gandhi Government General Hospital,Chennai-3 in partial fulfillment of the requirement for the award of the degree of Master of Surgery in General Surgery in February 2014, under my guidance and supervision. This dissertation is original and no part of this study has been submitted for the award of any other Degree or Diploma.

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DECLARATION

I, **Dr. M.ABDUL MALIQ,** solemnly declare that the dissertation titled **"THE PROSPECTIVE STUDY ON PREVALENCE OF HELICOBACTER PYLORI IN GASTRODUODENAL PERFORATION"** was done by me at the Rajiv Gandhi Government General Hospital, Chennai-3, under the guidance of **Prof. T.Bavani Sankar M.S.**

The dissertation is submitted in partial fulfillment of requirement for the award of M.S. Degree (Branch–I) in General Surgery to **The Tamil Nadu Dr.M.G.R. Medical University.**

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Place:

Date:

LIST OF ABBREVIATIONS

H. Pylori	→	Helicobacter Pylori
IVF	→	Intravenous Fluids
NSAIDs	→	Non-steroidal Anti-inflammatory Drugs
PPI	→	Proton Pump Inhibitor
Pts	→	Patients

ABSTRACT

Aim:

To study the prevalence of helicobacter pylori infection in Gastroduodenal perforations in Rajiv Gandhi Government General Hospital, Chennai .

Methods:

Prospective observational study. The study includes the patients admitted ,evaluated and diagnosed as Gastroduodenal perforation intra operatively. Biopsy was taken from the perforated ulcer site and adjacent mucosa, which was sent for Histopathological examination for Helicobacter Pylori detection by Giemsa staining.

Results:

50 patients presenting intra-operatively with Gastroduodenal perforation were studied for H.Pylori by histopathological examination. Of them, 44(88%)patients were found to have duodenal perforation and 6(12%) were found to have Gastric perforation. In the duodenal perforation group, 21(42%)patients were found to be H. pylori positive. In the gastric perforation group, 1(2%)patient was found to be H. pylori positive.

Keywords: Gastroduodenal perforations, Helicobacter pylori.

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PROFORMA

Name: I.P. No:

Age: DOA

Sex: DOS:

Address: DOD:

HISTORY

Chief Complaints:

Pain: Yes/No

Time of onset:_____

Mode of Onset_____ Site_____

Character _____Degree, Mild/Mod/ Severe,

Radiation _____, Aggravating / Relieving

factor _____ food – increase / decrease.

Vomiting: Yes / No

Time Of onset _____ Frequency, _____ Contents

_____ Projectile/non-projectile, relation to pain–increase /decrease

Distension of abdomen : Yes / No

Time of Onset _____ Relation to pain, - increase / decrease

Bowels:

Late evacuated _____ Constipation / Diarrhoea

FEVER –Yes / No

Duration _____ Degree _____ Chills / Rigors

H/O Drug intake _____

PAST HISTORY

Pain abdomen – Yes/ No Duration _____

Relation to food _____ Periodicity _____

H/O. – Hematemesis / Malena – T.B / Previous treatment / surgery

FAMILY HISTORY:_____

PERSONAL HISTORY:

Smoking / Alcoholism:

FOOD HABITS: Regular / Irregular

GENERAL PHYSICAL EXAMINATION

Appearance : Toxic / Non – Toxic, Pallor _____

Built / Nourishment: Poor / Mod /Well

Consciousness: _____

Hydration _____ Pulse _____ / min, B.P ___ mm of

Hg Temperature _____

PER ABDOMINAL EXAMINATION

Inspection:

Shape _____ Movt. with respiration _____Umbilicus

_____ scars of previous surgery _____Hernial orifices

_____

Palpation:

Tenderness: Site _____ guarding / rigidity – Yes / No

Rebound tenderness: Yes / No

Organomegaly: Yes / No Mass _____

Percussion:

Note over abdomen: _____

Obliteration of liver dullness Yes / No

Shifting dullness : - Yes / No

Auscultation

Bowel sounds: Present / absent Frequency:_____

Character _____

Per Rectal: _____

Per Vaginal : _____

OTHER SYSTEMS

CVS _____

RS _____

CNS _____

PROVISIONAL DIAGNOSIS

_____

INVESTIGATIONS

Hb _____ TC _____ DC_____ ESR _____

1^{st} hr

Urine albumin _____, Sugar, ____ Micro_____ RBS _____

HIV (spot) _____- HbsAg: _____ Sr. Electrolytes _____

S.Urea _____ Sr. Createnine _____

RADIOLOGICAL INVESTIGATIONS

X-ray abd/ erect / lateral decubitus: _____

Air under diaphragm: _____

Air fluid level: _____

Other findings: _____

Widal test: _____

Chest x-ray: _____

PREOPERATIVE TREATMENT

Antibiotics: _____

IVF: _____

Gastric aspiration: _____

Others: _____

OPERATIVE DETAILS

Incision:_____

Laprotomy findings:_____

Type of procedure:_____

Closure: Single layered / layered closure

POSTOPERATIVE MANAGEMENT

IVF _____ Antibiotics _____

H2 blocker _____

Blood transfusin _____ RTA _____

Oral feeds started on _____

Drain removal _____-

KEY TO THE MASTER CHART

IP No.	→	In patient Number
DOA:	→	Date of Admission
DOD	→	Date of Discharge
DOP	→	Date of Operation
BS	→	Bowel Sound
LAP and Graham's Patch	→	Laparotomy and Graham's Patch
TLC	→	Total Leucocyte Count
DLC	→	Differential Leucocyte Count
Y	→	Yes
N	→	No
A	→	Absent
P	→	Present

INTRODUCTION

Gastro duodenal ulcer is one of the most common malady that affects the mankind in India. Though lot of work had been done on the etiology of this condition, one specific etiological agent cannot be incriminated in the causation of this particular disease especially in our part of country. Since, stress forms the most important single feature in causing peptic ulcer and today's modern life is full of stress and strain, this condition on the increase.[1]

Perforation of gastric or duodenal ulcer is one of the most serious and most overwhelming catastrophic event that is affecting human being (Lord Moynihan).[2]

Among abdominal emergencies, perforations of peptic ulcer are third in frequency, acute appendicitis and acute intestinal obstruction being more common. Prompt recognition of the condition is very important and only by early diagnosis and treatment it is possible to reduce the still relatively high mortality.[3]

There is decline in incidence of duodenal ulcers and elective surgery for duodenal ulcers, which is attributed to the era of H2 blockers and proton pump inhibitors, which provides symptomatic relief to patient. But the percentage of patients with perforation has not declined, probably due to increased inadvertent use of NSAIDs, corticosteroids and because of irregular use of H2 antagonist drugs.

The treatment of perforation still continues to be controversial. Just closure of perforation may save life, but chance of recurrence of ulcer is too high and patient may not turn up for a second curative surgery. So, there is a school of thought, which recommends definitive surgery in a perforated peptic ulcer. This may to a certain extent reduce the mortality and morbidity of the patient, because patients have to risk major operation when the general condition is not good. On the other hand it saves the patient of further surgery.

When acute or chronic duodenal ulcer perforates into the peritoneal cavity, three components require treatment viz., the ulcer, the perforation and the resultant peritonitis. The perforation and resultant peritonitis are immediate threats to the life, the ulcer in itself is not. The theraptic

priorities thus are treatment of peritonitis and securing the closure of perforation, which may be achieved with surgical procedure.[5]

Inspite of better understanding of disease, effective resuscitation and prompt surgery under modern anesthesia techniques, there is high morbidity (36%) and mortality (6%).

AIMS AND OBJECTIVES

To study the prevalence of helicobacter pylori infection in Gastroduodenal perforations in Rajiv Gandhi Government General Hospital, Chennai .

REVIEW OF LITERATURE

HISTORICAL REVIEW[6,7]

Surgeons have attempted for 100 years to cure the duodenal ulcer by reducing the secretion of acid and pepsin, and history of surgery for peptic ulcer is a chronicle of their attempts to achieve this aim without producing major disturbance to the functions of alimentary tract. Perforated peptic ulcers, as a disease entity has been known since 1660.

1660: Littre, England first described gastric ulcer perforation as the cause of death of daughter of Charles-I of England.

1726: George Hamberg, Germany described a duodenal ulcer.

1727: Christopher Rawlinson, England first described a case of perforated peptic ulcer.

1793: Jacopo Penada, Italy first recorded a duodenal perforation.

1881: Ludwig Rydygier, performed a successful resection of a prepyloric peptic ulcer.

1881: Theodor Billroth, Father of Surgical Audit and Father of Abdominal surgery, performed the excision of distal part of the stomach with an anastomosis of the gastric stump to the duodenum (Billroth I Surgery).

1886: Heineke, did the first pyloroplasty.

1888: Mikulicz redefined the pyloroplasty done by Heineke.

1893: Barling, of Great Britain, treated perforated ulcer by closure and vigorous lavage of peritoneal cavity with large quantity of saline.

1893: Codivilla reportedly did the first gastrojejunostomy for a duodenal ulcer.

1896: Bennett suggested sealing a large perforation with omentum[5]

1899: Kently performed gastric resection for perforated pepticulcer.

1937: Cellian-Jones and Graham popularized the effectiveness of omental patch for perforation.

1943: Dragsted and Owens introduced bilateral truncal vagotomy.

1948: Franksson of Stockholm first reported selective vagotomy.

1965: Erik Amdrup performed highly selective vagotomy.

1970: Robin Warren reported an association between Helicobacter pylori, gastritis and peptic ulcer perforation.

1985: Barry Marshall cultured Helicobacter pylori.

1985: Johansson B. Gilse H. described a laparoscopic technique for closure of perforated peptic ulcer.

1996: Halkic N. Pescatore P. and Gilleton combined both laproscopic – endoscopic method using an omental plug for therapy gastroduodenal ulcer perforation.

Perforation of peptic ulcer is now a common complication, second to penetration. It was rare until the end of the 19th century, but since then its frequency has increased progressively. Moreover, there was a curious change in incidence in the 19th century, most perforations were gastric perforations and the majority affected women, especially girls aged from 10-28 years. By 1959, Duodenal perforations greatly exceeded gastric,men were affected more than women and most cases occurring between 25-45 years.

ANATOMY OF STOMACH[8,9]

Embryology :

During the fifth week of gestation the stomach arises as a dilatation in the tubular embryonic foregut. It assumes its normal asymmetric shape and position by the end of the seventh week through descent, rotation, and progressive dilation, with disproportionate elongation of the greater curvature. It is likely that there is a congenital predisposition to some unusual benign gastric problems such as diverticulum or massive hiatal hernia with abnormal gastric rotation and fixation.

Parts & Relations of Stomach :

The stomach is divided by arbitrary lines drawn on its external surface into a fundus, body, pyloric antrum and pylorus (Fig.1). The internal appearance and microstructure of these regions varies to some degree. The fundus is dome shaped and projects above and to the left of the cardiac orifice to lie in contact with the left dome of the diaphragm. It lies above a line drawn horizontally from the incisura cardiaca to the greater curvature. The body extends from the fundus to the incisura angularis, which is a constant external notch at the lower end of the lesser curvature. A line drawn from the incisura angularis to an indentation on

the greater curvature defines the lower boundary of the body. The pyloric antrum extends from this line to the sulcus intermedius, where the stomach narrows to become the pyloric canal (1–2 cm long), which terminates at the pyloric orifice.

The organs in relations to stomach are the liver, colon, spleen, pancreas, and occasionally the kidney (Fig.1). The left lateral segment of the liver usually covers a large part of the anterior stomach. Inferiorly, the stomach is attached to the transverse colon by the gastrocolic omentum. The lesser curvature is tethered to the liver by the hepatogastric ligament, also referred to as the lesser omentum. Posterior to the stomach is the lesser omental bursa and the pancreas.

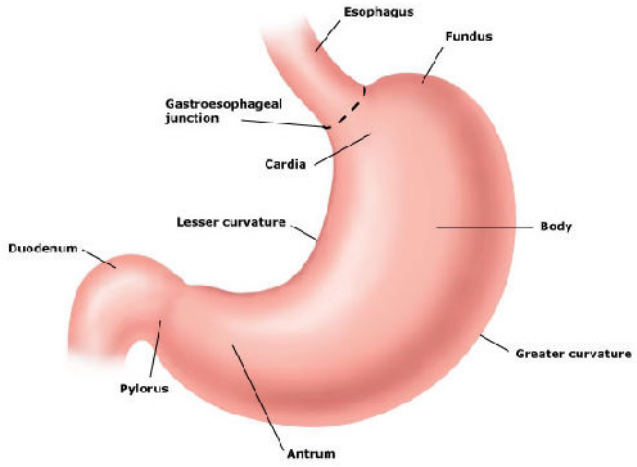

Fig 1. Parts of Stomach

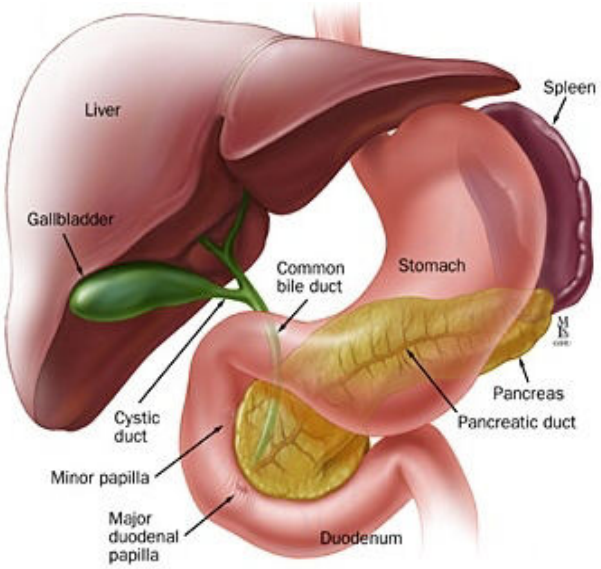

Fig 2. Relations of Stomach

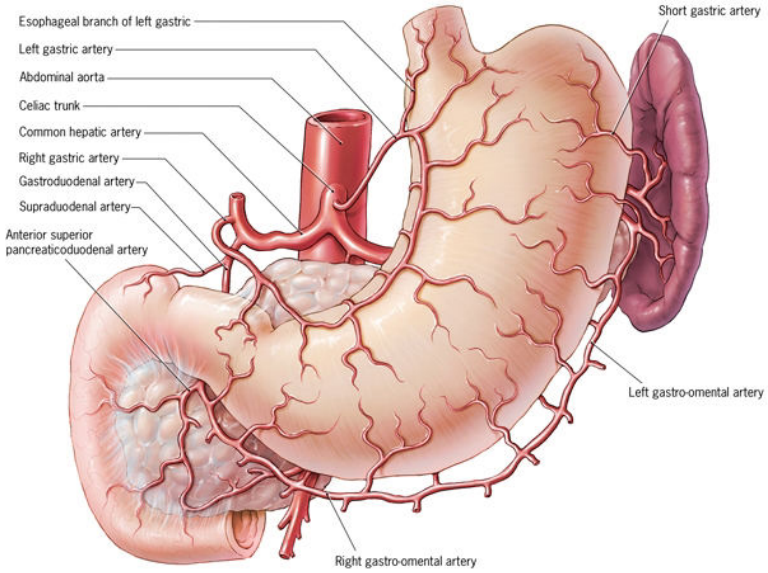

Fig. 3 Arterial supply of Stomach

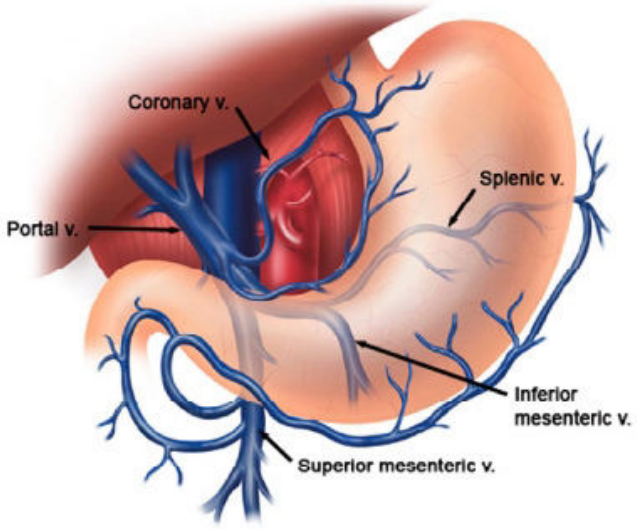

Fig 4. Venous drainage of Stomach

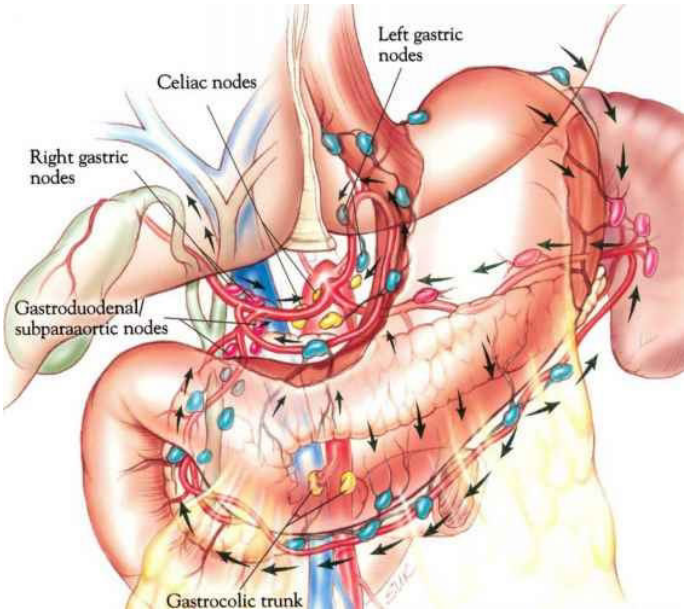

Fig 5.Lymphatic drainage of Stomach

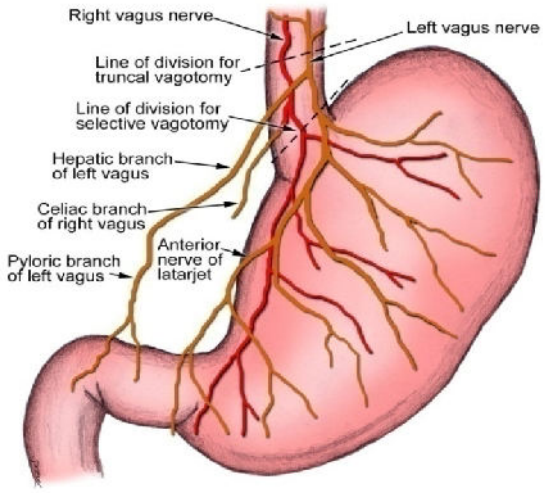

Fig 6.Nerve Supply of Stomach

Artery Supply of Stomach :

The arterial supply to the stomach comes predominantly from them coeliac axis although intramural anastomoses exist with vessels of other origins at the two ends of the stomach (Fig.2). The left gastric artery arises directly from the coeliac axis. The splenic artery gives origin to the short gastric arteries as well as the left gastroepiploic artery and may occasionally give origin to a posterior gastric artery. The hepatic artery gives origin to the right gastric artery and to the gastroduodenal artery, which in turn gives origin to the right gastroepiploic artery.

Venous drainage of Stomach :

The veins draining the stomach generally parallel the arteries. The left gastric (coronary vein) and right gastric veins usually drain into the portal vein, though occasionally the coronary vein drains into the splenic vein. The right gastroepiploic vein drains into the superior mesenteric vein near the inferior border of the pancreatic neck, and the left gastroepiploic vein drains into the splenic vein.

Lymphatic drainage :

The stomach has a rich network of lymphatics (fig.4) that connect with lymphatics draining the other visceral organs of the upper abdomen. At the gastro-oesophageal junction the lymphatics are continuous with those draining the lower oesophagus, and in the region of the pylorus they are continuous with those draining the duodenum. In general, they follow the course of the arteries supplying the stomach. However, many separate groups of nodes are now recognized, and their relationship to the regions of the stomach and the vascular territories supplied, is of great importance during resection of the stomach, particularly for malignancy. Pancreatic and hepatic lymphatics play a significant role in draining areas of the stomach during disease.

Nerve Supply of Stomach :

The stomach is supplied by sympathetic and parasympathetic nerves. The sympathetic supply to the stomach originates from the fifth to 12th thoracic spinal segments, and is mainly distributed to the stomach via the greater and lesser splanchnic nerves via the coeliac plexus. Periarterial plexuses form along the arteries and supply the stomach from the coeliac axis. Additional innervation comes from fibres of the hepatic plexus, which pass to the upper body and fundus via the upper limit of the lesser omentum and via direct branches from the greater splanchnic nerves.

The parasympathetic supply to the stomach is from the anterior and posterior vagus nerves (fig). The anterior vagus (formed mainly from fibres from the left vagus originating from the oesophageal plexuses) is often double or even triple and supplies filaments to the cardiac orifice. The posterior nerve tends to lie more medial than the anterior and is often a short distance from the wall of the abdominal oesophagus. The parasympathetic gastric supply is secretomotor to the gastric mucosa and motor to the gastric musculature. It is responsible for coordinated relaxation of the pyloric sphincter during gastric emptying.

ANATOMY OF DUODENUM[8,9]

EMBRYOLOGY [8,9] :

The superficial (1st) part and the upper half of the descending (2nd) part of duodenum are derived from the foregut. The rest of the duodenum develops from the cephalic part of midgut. The junction of which is located directly distal to the origin of liver bud.

As stomach rotates, the duodenum takes on the form of C-shaped loop and rotated to right. This rotation and rapid growth of the head of pancreas causes duodenum to swing from its initial midline position to right side of abdominal cavity. The duodenum gets attached to dorsal wall of abdomen by a mesentery called mesoduodenum. Later this mesoduodenum fuses with peritoneum of the posterior abdominal wall causing most of duodenum retroperitoneal, except in the region of the pylorus of stomach, where a small portion of duodenum remains intraperitoneal, which is seen as **duodenal cap** in radiographs.

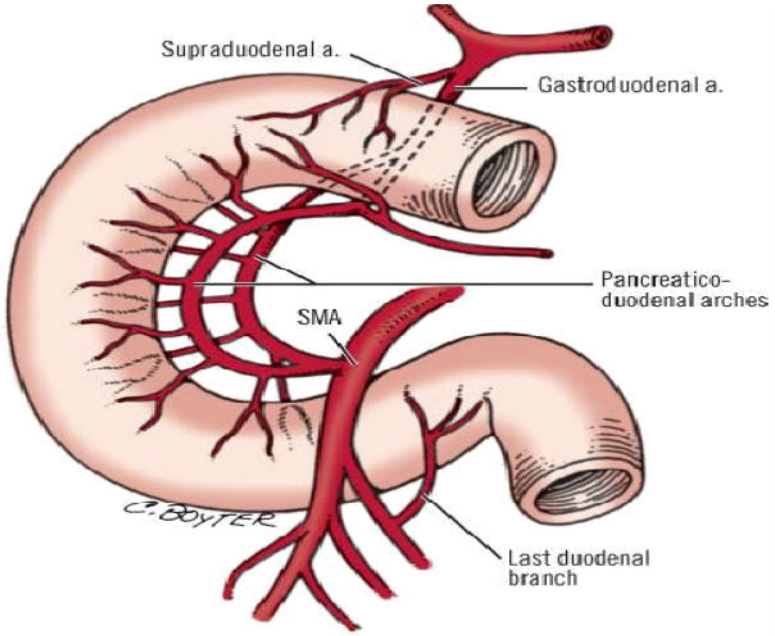

Fig. 7: Arterial Supply of duodenum

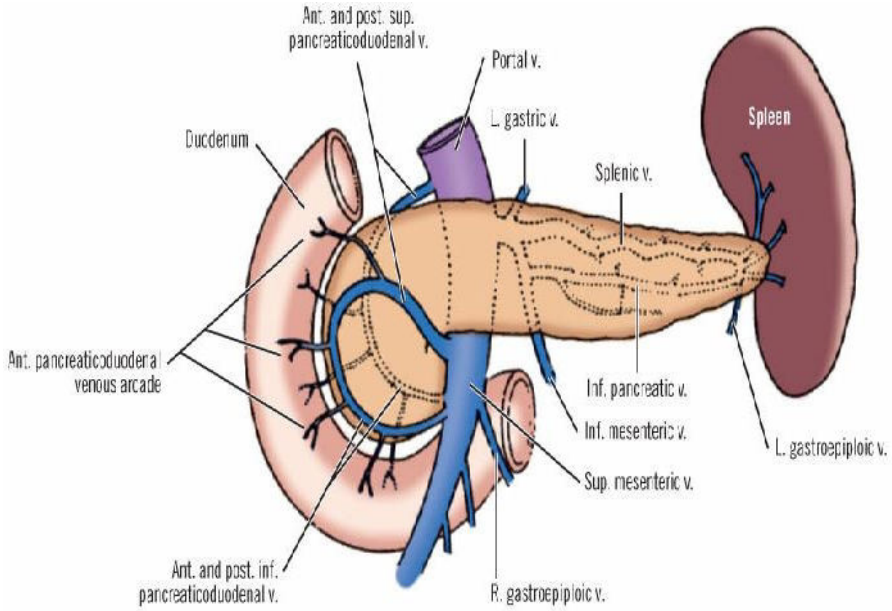

Fig 8. Venous drainage of duodenum

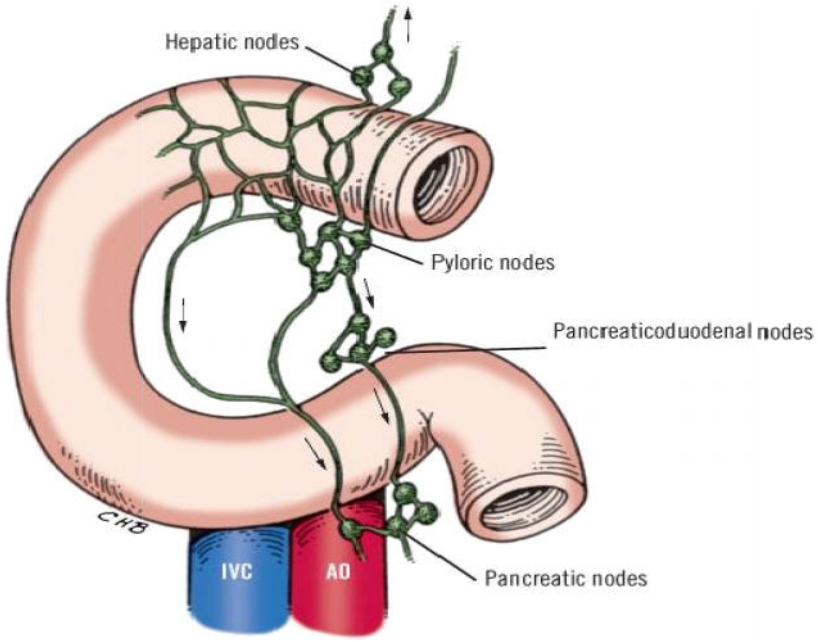

Fig. 9: Lymphatic drainage of duodenum

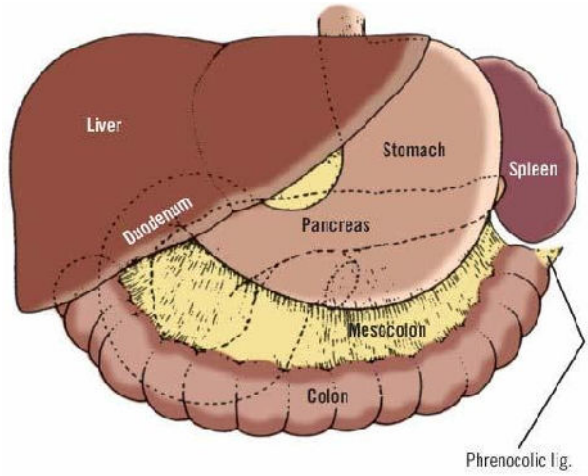

Fig. 10: Relations of duodenum

PARTS & RELATIONS OF DUODENUM

The duodenum is a C-shaped, first and shortest (about 10inches/25cms and most fixed part of the small intestine. It has no mesentery and thus is only partially covered with peritoneum. It extends from the pylorus to the duodenojejunal junction, making C-shaped curve, which is occupied by the head of pancreas and lies entirely above the level of umbilicus.

Parts of duodenum: The duodenum is situated in the epigastric and umbilical regions and is divided into **four** parts.

I. **First/Superior part of the duodenum:** It is 2inches/5cms long, it begins at the pylorus and runs upwards and backwards on the right side of the first lumbar vertebra towards liver and ends at the neck of gallbladder by bending sharply. It thus lies on the transpyloric plane. The first inch is covered with peritoneum on the front and back and can be moved with the stomach. The second inch is covered with peritoneum only above and in front.

II. **Second/Descending part of the duodenum:** It is 3inches/8cms long. It runs vertically downwards in front of the hilum of the right kidney on the right side of the L2 and L3 vertebrae. It is crossed by the transverse colon. About halfway down on its medial border, the bile duct and the pancreatic duct unite to form a short dilated tube called hepatopancreatic ampulla, narrow distal end of this opens on the summit of the major duodenal papilla. The accessory pancreatic duct when present opens 2cms proximal to the major duodenal papilla as minor duodenal papilla.

III. **Third/Horizontal part of the duodenum:** It is 3inches / 8cms long. It runs horizontally and to the left on the subcostal plane and is crossed by the root of the mesentery.

IV. **Fourth/Ascending part of duodenum:** It is 2inches / 5cms long, shortest part of the duodenum. It runs upwards along the left side of the aorta on the left psoas muscle and ends about an inch to the left of the median plane at the level of the L1 vertebra. The duodenojejunal flexure is usually retroperitoneal, lies to the left of the disc of L1 & L2 vertebrae. It is fixed and held in position by the peritoneal fold called Ligament of Treitz, which is attached to the right crus of diaphragm.

Blood supply

Arterial supply: The upper half of the duodenum is supplied by the superior pancreatoduodenal artery, a branch of gastroduodenal artery. The lower half is supplied by inferior pancreatoduodenal artery a branch of the superior mesenteric artery.

Veins: Drains to superior mesenteric and portal veins.

Lymphatics: The lymph vessels follow the artery and drains upward via pancreatoduodenal nodes to the gastroduodenal nodes and to the coeliac nodes: and downward via pancreatoduodenal nodes to superior mesenteric nodes.

Nerve supply is derived from the sympathetic and parasympathetic (vagus) from the coeliac and superior mesenteric plexus.

PHYSIOLOGY[10,11]

The various functions of stomach are:

1. It begins the process of food breakdown exposing solid meal to proteolytic action of acid and pepsin.
2. It grinds and dilutes the mixture to form a more uniform consistent chyme.
3. It acts as a reservoir when food is stored for a period of approximately 4 hours.

GASTRIC SECRETION

The stomach secretes water and electrolytes, primarily in the form of acid and small amount of bicarbonates, enzymes such as pepsin, glycoprotein such as intrinsic factor and mucous. Gastric juice also contains small amounts of calcium, magnesium and trace amount of zinc, copper and iron.

ACID SECRETION

Human stomach secretes about 2-5 mEq/hour of HCL in the fasting state, constituting basal acid secretion. After a mixed meal, the amount of acid secretion increases to 1 5-25mEq/hour. Acid is secreted by parietal cells situated in the glands of the fundus and body of the stomach. Regulation of acid secretion is a complex process involving endocrine, neural, paracrine and even autocrine mechanisms.

There are three phases in gastric secretions-

- Cephalic phase: Is stimulated by the sight of smell of chewing of food.
- Gastric phase: Is stimulated by the presence of food in the stomach
- Intestinal phase: Is stimulated by the presence of food in the small intestine.

1. **Cephalic phase:** Cephalic phase stimuli (sight or smell of food) presumably activate the vagal nuclei in the medulla. Impulses traverse the peripheral vagi and terminate in the gastric mucosa with the release of acetylcholine from vagal nerve endings. Release of acetylcholine in the fundic mucosa directly, stimulates and secretion by the parietal cell and release of pepsinogen by chief cells. Acetylcholine release in the antral mucosa may cause discharge of the antral hormone gastrin. Distensi

2. on of stomach excites vaso-vagal reflex that also results in the release of acetylcholine in the fundic and antral mucosa.

3. **Gastric phase:** This phase is initiated by the entry of food into the stomach. Food that enters the stomach buffers acid, raises pH and allows other stimuli to release acid. Through this gastrin is liberated from the gastric mucosa either due to antral distension and when the pH reaches 1.5, gastrin output is absolutely stopped. So this is a feedback mechanism in which production of gastrin is inhibited by the presence of acid in the antrum of stomach. The most remarkable action of gastrin is its ability to stimulate gastric

acid secretion. It is 30 times more potent than histamine. Beside its action on acid secretion, it stimulates pepsin secretion and increases gastric mucosal blood flow. It also stimulates pancreatic enzyme secretion in man.

4. **Intestinal phase:** The intestinal phase of secretion begins as chyme begins to empty from the stomach into the duodenum. Distension of jejunum will also stimulate secretion. The cholecystokinin, the duodenal hormone which acts to stimulate secretion of pancreatic enzymes an stimulate contraction of gall bladder, also acts like gastrin

INHIBITION OF GASTRIC SECRETION[11]

Once cephalic stimulation is removed vagal activity is decreased. Most important is the secretion of acid itself blocks the further release of gastrin and to bring about active duodenal suppression of gastric secretion.

Antral acidification has been clearly demonstrated to suppress the release of gastrin. Significant diminution in acid stimulation may occur with an antral pH as high as 5 and at pH 1.5 there is no release of gastrin. Antral inhibition is apparently due to a passive removal of the gastric

stimulus but there is clear evidence of active mechanisms of duodenal inhibition. Gastric secretion is inhibited by the presence of acid or fat or hypertonic solution in the duodenum. For a time gastric inhibitory polypeptide (GIP) appeared to be responsible for this enterogastrin like activity but recent evidence suggests that GIP is a weak inhibitor of gastric acid secretion in man and that its chief function is likely to be that of glucose dependent releaser of insulin. Whether nervous reflexes play a primary or a permissive role in duodenal inhibition has not been clarified. Acidification of duodenum inhibits gastric secretion. It also releases secretin and secretin is known to inhibit gastrin stimulated gastric secretion.

PEPTIC ULCER DISEASES[12-16]

Peptic ulcer is a term used to refer to a group of ulcerative disorders of the gastrointestinal tract, involving principally the most proximal position of duodenum, the stomach, the lower end of the oesophagus, the jejunum after surgical anastomosis to stomach or rarely the ileum adjacent to the Meckel's diverticulum due to ectopic gastric epithelium.

Approximately 98-99% of peptic ulcers occur in the first portion of duodenum or in the stomach, in a ratio of about 4%. About 5% of individuals with gastric ulcer develop duodenal ulcers, but 20% of those with duodenal ulcers develop gastric lesions.

The pyloric channel, which is 1-2cms in length, is the narrowest portion of the gastric outlet. Because of their gastric acid secretory characteristics and clinical features, pyloric channel are classified with duodenal rather than gastric ulcer. Ulcers in this location often produce symptoms similar to those of duodenal ulcer. In-patients with pyloric channel ulcers, food may accentuate rather than relive ulcer pain.

EPIDEMIOLOGY

Peptic ulcers are remitting relapsing lesions, at one time duodenal ulcers were much more common than gastric ulcer, but their incidence and prevalence are now approaching those of gastric ulcers.

Most often diagnosed in middle aged to older adults, but may first become evidence in young adult life. Male to female ratio for duodenal ulcer is about 3:1 and for gastric ulcers around 1.5:2.1. Women are most often affected at or after the menopause.

Genetic influence plays some role in predisposition to both forms of ulcers, but more clear cut with the duodenal ulcers. Duodenal ulcers are three times more common in the first-degree relatives of ulcer patients than in general population. A 50% concurrence of duodenal ulcers in monozygotic twins, but only 14% in dizygotic twins.

An increased incidence of HLA-B5 antigen has also been identified in white males with duodenal ulcers.

Individuals with blood group 'O' are about 30% more likely to develop duodenal ulcer than those with other blood group.

Increased use of NSAIDs and corticosteroids in one of the common cause in producing duodenal ulcer.

PEPSIN SECRETION: It is influenced by hydrogen ion secretion, cholinergic stimuli and by polypeptide hormones. Increased H+ secretion causes increased pepsin secretion.

GASTRIC MUCUS SECRETION: This is secreted by gastric mucosa serves the function of the lubrication, protecting the mucosa from mechanical damage and gastric acids. Its secretion is evoked by vagal stimulation, on feeding and gastric irritation. Its pH is alkaline, the

mucous barrier is damaged by bile acids refluxed through the pylorus and drugs such as salicylates and alcohol.

Duodenal Exocrine Secretion: It is alkaline in nature, secreted by Brunner's gland into the crypts of Lieberkuhn, the amount of secretion is related to the acid delivered through the pylorus.

GAST

RIC AND DUODENAL ENDOCRINE SECRETION

From the Stomach: Gastrin is secreted by the 'G' cells of the antrum, exists as G-17 little big gastrin predominantly found in circulation.

Functions

- Stimulates HCl secretions from the parietal cells. Pepsinogen is converted into pepsin in the presence of HCl.
- Stimulates bile secretion from the liver and promotes gall bladder contraction.
- Stimulates pancreatic enzymes and bicarbonate secretion.
- Gastrin secretion is inhibited by fall of gastric pH below 3 and by somatostatin.

From the Duodenum:

The presence of acid chyme in the duodenum stimulates the secretion of secretin, Cholecystokinin-pancreozymin (CCK-PZ), enteroglucagon and enterogastrone hormones.

Functions of secretin

1. Inhibits acid secretion from parietal cells.
2. Stimulates bile secretion from the liver.
3. Stimulate bicarbonate secretion.

Environmental factors: Duodenal ulcer is more frequent in patients with alcoholic cirrhosis, chronic renal failure, chronic obstructive pulmonary disease and hyperparathyroidism.

Role of Helicobacter Pylori Infection in peptic ulcer

The word "No acid-No ulcer" does not holds good now a days, because peptic ulcer is considered now more an infective disease, caused by Helicobacter Pylori.[14]

In 1983, Warren and Marshall first reported isolation of Helicobacter Pylori from the mucosal biopsy specimen of patient with peptic ulcer diseases.[11]

Helicobacter pylori are a small spirally curved, gram negative, microaerophilic rod with multiple pollar flagellae. 80–90% of populations are affected with infection of Helicobacter pylori. The incidence of infection within a population increases with age. The possibility of infection is inversely related to socioeconomic group.[16]

Helicobacter pylori infection is the major cause of peptic ulcer not associated with the use of non-steroidal anti-inflammatory drugs. Human are the major reservoirs of Helicobacter pylori. The organism colonizes in the stomach, lodging most frequently in the antrum. The route of transmission of Helicobacter pylori infection is mainly by faeco-oral route and oro-oral route.[17]

Pathogenesis of Gastroduodenal Ulcer due to Helicobacter Pylori[13]

Helicobacter pylori infection invariably results in chronic gastritis. The clinical result of this infection ranges from asymptomatic gastritis to peptic ulceration and gastric cancer. Helicobacter pylori colonizes in the gastric epithelium causing Type-B gastritis by which it reduces the resistance of gastric mucosa to attack by acid and pepsin resulting in gastric ulcer. Although, Helicobacter pylori normally reside in stomach, it also leads to causation of duodenal ulcers. This can be explained by the

fact that antral Helicobacter pylori infection impairs the inhibitory feedback control of acid secretion, thus promoting duodenal ulcerogenesis by increasing duodenal acid load, resulting into duodinitis which leads to local inflammation, mucosal injury and eventually ulcer formation through the following mechanism:

- By increasing acid secretion: One of the characteristic features of the organism is the production of urease enzymes, which hydrolyzes urea, resulting in production of ammonia, a strong alkali. Ammonia generated causes the release of gastrin (hypergastrinaemia) from antral G-Cells, which in turn leads to gastric acid hypersecretion.
- By disrupting gastric mucous barrier.
- By secretion of number of enzymes and chemicals, urease, catalase, mucin, lipase, phospholipase, porins, protease's, hemolysins and alkaline phosphatase.
- By inducing inflammation in gastric migration and degeneration of acute inflammatory cells, such as neutrophils and accumulation of chronic inflammatory cells, such as macrophages and lymphocytes.

Overall, Helicobacter pylori are undoubtedly the dominant factor in the pathogenesis of peptic ulcer disease. There is, however, a small minority of duodenal ulcers where Helicobacter pylori has no effect, such as ulcers related to use of NSAIDs, Crohn's disease and in Zolinger EllisonSyndrome.

Helicobacter pylori infection has also been implicated as a risk factor for gastric carcinoma and low-grade gastric lymphoma of mucosa associated with lymphoid epithelium (Wyatt and Dixon): WHO has described Helicobacter pylori as Class-I carcinogen.

PATHOGENESIS OF PEPTIC ULCER[13]

All peptic ulceration probably arises because of an imbalance between the aggressive action of acid pepsin secretion and the normal defenses of the gastroduodenal mucosa.

For duodenal ulcer, the major causal influence appears to be exposure of the duodenal mucosa to excess amount of acid and pepsin.

For gastric ulcer, the major causal influence appears to be some breakdown in the gastric mucosal defenses against acid and pepsin.

The hypersecretion is related to an abnormally large total mass of parietal cells in the gastric mucosa, perhaps to either increased

responsiveness of the parietal cells to secretory stimuli or lack of normal regulatory controls.

Increased levels of gastric or unusual sensitivity of the parietal cells to gastrin stimulation may also be involved.

Individual with total achlorhydria never develops a duodenal ulcer.

Defect in the defense mechanism includes deficiencies in mucosal cell removal, in mucous production in elaboration of bicarbonate and in production of prostaglandin.

Irrespective of treatment, peptic ulcer takes one of the courses during the period of its progress:

- Healing
- Chronicity
- Complications

The **complications** of peptic ulcer are:

1. Haemorrhage
2. Perforation
3. Cicatrical contraction
4. Carcinomatous changes

ETIOLOGY[18-21]

Familial and genetic factors: Duodenal ulcer is 2 to 3 times more prevalent in relatives of patients with ulcer, as supported by family studies and genetic marker investigations.

Cultural and social factors: Emotions, stress, cultural and social factors are involved in the course of peptic ulcer disease.

Cigarette smoking: The effect of cigarette is claimed to be dose dependent. The smoking of cigarette inhibits the synthesis of prostaglandins, reduces mucosal blood flow, and inhibits pancreatic bicarbonate secretion. Also, acid secretion following stimulation by a gastrin analogue is greater in smokers. Cigarette smoking adversely affects ulcer healing and worsens prognosis.

Dietary factors: Alcohol can cause mucosal damage .But prohibition of alcohol use does not have a documented benefit. Caffiene also may increase gastric acid secretion.

Non-steroidal Anti-inflammatory Drugs: Non-steroidal anti-inflammatory drugs are frequent cause of gastric and duodenal mucosal injury. They impair defence mechanisms by decreasing mucosal and bicarbonate secretion, changing prostaglandin output, and altering other physiologic and anatomic factors.

Adrenocorticoids: High dose adrenocorticoids have been implicated in some ulcerations of the mucosa of upper GIT by inhibiting regeneration of rapidly dividing cells of the gastrointestinal mucosa**.**

Helicobacter pylori: H. pylori infection of the antral mucosa results in depletion of antral somastatin and increased release of gastrin and thus increased acid secretion in patients with duodenal ulcer disease. Increased acidity damages duodenal mucosa, thereby resulting in the development of gastric metaplasia .Such damaged mucosa is then colonized by H. pylori and the combination of acid and infection leads to formation of duodenal ulcer disease. More than 95% of patients with chronic recurrent duodenal ulceration have this infection and eradicating it considerably reduces the ulcer recurrence rate.

Association with other disease: There is thought to be an association between chronic lung disease, chronic renal failure and cirrhosis {these diseases often involve use of cigarettes, analgesics and alcohol}. Patients with rheumatoid arthritis probably have propensity for ulcer disease, which is independent of use of NSAIDs**.**

Age: Till 1940, 75% of perforations occurred in third to fifth decades. But since then there has been increasing percentage of

perforation in sixth to eighth decades. No age is exempted but rare in childhood. Perforation rarely occurs during neonatal period and early childhood. Ream (1963), reported a mortality of 57% in 39 neonatal perforations. Perforations were uncommon in adolescent. Mohammed and Mackey(1982), described 22 patients of peptic ulcer, out of which only 3 were perforations (13.6%). A study by J. Higham and colleague in Great Britain (1989-1999), shows perforations were highest in patients more than 65yrs of age.

Sex: Perforation more common in male than women. A study by J. Higham and colleagues from Great Britain (1989-1999), reported that perforation from gastric ulcer declined but perforation from duodenal increased among men at older age.Mc Kay and Mc kay (1976) reported the following male : female ratio 4.1:1

Occupational Incidence: The perforations are more likely to occur in those engaged in heavy manual work. Kozal and Mayer (1960) reported population incidence in 1904 perforations is as follows:

- Unskilled: 27.9%
- Semiskilled: 14.5%
- Skilled: 12.9%
- Dependents: 11.0%

Hence, perforation is highest in semiskilled or unskilled workers.

Daily Variation: Jamioson in 1944, Luer in 1949, Spence in 1950 found increased incidence of perforations in afternoons and evenings and less incidence in the nights. Hennessy in 1969 and Hendry and colleagues in 1984, suggest that there are two peaks of incidence, at the beginning of the day and in the evening, indicating that again periods of stress and strain are predisposing factors for perforations.

Relation to Meals: Jamioson (1944), Bean (1943), stated perforation is more 2-3hrs after meals, which may be due to over distension of stomach. Dr. S.S Hussain (1965) say perforation is more common immediately after food.

Helicobacter pylori and Perforation: In recent years, even though Helicobacter pylori is implicated as main causative organism for peptic ulceration, its direct relation to perforation is not proved yet.

Medical Treatment: Irregular use of H2 receptor antagonist Cimetidine: was associated with perforation. Wallase (1977) reported three cases of perforation of chronic peptic ulcer, in which abrupt cessation of treatment precipitated perforation. Those perforations may be due to acid rebound.

Pregnancy and Perforation: Wary (1945) noted increased incidence of perforation in pregnant women and presumed that hormonal basis for its cause.

Relation of Physical Stress: DeBakey in 1940 says trauma (in 4% of patients) plays role in perforation, whereas Jamieson in 1944 says that severe exertion plays little part in perforation of peptic ulcer.

Other predisposing factors: Upper respiratory tract infection, Fatigue, exposure to cold damp weather, worry and anxiety, alcoholism, heavy smoking and failure to maintain control of diet are some contributing factors during period of exacerbation.

PATHOLOGICAL COURSE[12,13]

At the onset of perforation there is sudden spillage of duodenal contents into the general peritoneal cavity and results in chemical peritonitis. The degree of involvement of the peritoneal cavity by bacteria is always uncertain. It is suggested that at first the visceral contents are sterile and the infective peritonitis in the early case is unlikely. However, it depends on the general condition of the patient and his resistance to infection.

Perforation of duodenal ulcer may be classified as follows:

1. Acute perforation
2. Subacute perforation
3. Chronic perforation
4. Perforation associated with haemorrhage

1. **Acute perforation**

The ulcer perforate and the general peritoneal cavity become flooded

with gastric and duodenal contents, causing chemical peritonitis.

The clinical features vary according to the stage of perforation. The clinical course can be divided into three stages, each of variable duration:

1. Primary stage or the stage of peritonism.
2. Secondary stage or the stage of peritoneal reaction.
3. Tertiary stage or the stage of bacterial peritonitis.

a) **Primary stage:** The clinical course of a perforation is generally unmistakable. At that moment, the patient feels acute agonizing pain in the epigastrium or right hypochondrium, which usually becomes rapidly generalized. He is plunged into a state of prostration and may be rendered immobile and helpless.

The symptoms, which arise with dramatic suddenness, are due to the intense irritation of peritoneum by the gastric and duodenal contents. They produce neurogenic shock, in the early stages nausea and vomiting are uncommon. Abdominal pain, pain referred to the both shoulders as a result of irritation of diaphragm, IT IS Also Associated with subnormal temperature, cold extremities, pale facies and sweating. Expression is one of anxiety or fear. Patient lies almost rigid, with his legs drawn up and his hands held tensely to his side. The temperature may be subnormal, as low as 95^0 to 96^0 or normal. Pulse rate is normal or raised to 90mt or above Respiration is shallow with increased respiration rate. On inspection, the abdomen will be seen to be immobile and no movement with respiration. Rectus muscle shown into prominence, the muscles are rigid and board like. The rigidity is generalized as well as tenderness. On auscultation bowel sounds are absent. This stage lasts for 3-6 hours.

Sometimes the fluid creeping from the perforation may tickle down the paracolic gutter, producing signs suggestive of acute appendicitis, with tenderness and rigidity limited to the right side of the abdomen.

b) **Secondary stage:** Transition of the primary stage to secondary stage takes hours, depending on the size and site of perforation and amount of peritoneal soiling. It is during this stage the spontaneous

sealing of perforation may occur. If there is gross leakage of gastric contents, the patient may pass onto the stage of septic peritonitis. The length of this stage rarely exceeds 6 hours. During this stage the pain is lessened markedly. There would be general improvement in the patients' condition. For this reason this stage of reaction has sometime been called stage of delusion.

The improvement in the well being may cause the patient to delay calling medical attention, and it is in this stage most of the error in diagnosis takes place. On examination there will be varying amount of rigidity of abdomen and tenderness. Bowel sounds are infrequently heard or absent.

c) **Tertiary Stage:** This is the stage of diffuse peritonitis, begins about 12 hours after perforation and lasts for about 24 hours until it passes on to final stage of paralytic intestinal obstruction. Pathogenic organism multiplies rapidly. Peritoneal fluid becomes more purulent. The intestines slowly and pragmatically distend with gas and fluid. Intestinal movements diminish and finally disappear with onset of paralytic ileus. The clinical features are same as those of a generalized peritonitis from any other cause.

Pain is less severe, vomiting frequently, hiccoughs may further epress the patient. Sweating and vomiting and outpouring of fluid into peritoneal cavity, distended paralyzed intestine, dehydration and electrolyte imbalance become more evident. Patient complains of severe thirst, raised temperature, tongue,dry and coated, pulse thready, respiration is shallow and rapid. Abdomen distended, guarding still present. On auscultation. Bowelsounds absent. The typical Hippocratic facies denotes that end is not too for off. The face is ashen, body cold and clammy. The patient drifts into toxemia, dehydration and circulatory failure. Death usually takes place 4-5 days after perforation.

2. Subacute Perforation

An ulcer may perforate and the perforation may seal rapidly before there is spillage of gastric and duodenal contents, into general peritoneal cavity. There is sudden onset of acute abdominal pain often more severe to the right upper quadrant. Respiration will be shallow and deep inspiration may be associated with an abrupt catch in the breath.

On examination, there is local tenderness and rigidity, but rest of the abdomen will be “soft” to palpate and non-tender. Unusually and X-ray film reveal a small amount of gas under the diaphragm. After an hour or two, with bed rest, the pain will usually subside. Rarely tenderness and

rigidity may extend and the signs of an acute perforation develop.

3. Chronic Perforation

When an ulcer perforates into an area that is walled off by adhesions or by adjacent viscera such as liver, colon or greater omentum or when gastric ulcer perforates into omental sac, a chronic abscess may develop and will give rise to considerable confusion in diagnosis. As these patients do not present with signs and symptoms of peritonitis, they are seldom diagnosed as having perforated peptic ulcer. Irregular temperature, rigors, leucocytosis, dullness at the base of the lung, consequent pleural effusion or basal congestion will lead to the diagnosis of subphrenic abscess. An X-ray of abdomen may show subphrenic abscess, containing gas, and diaphragm is raised and fixed on right side. USG of abdomen is the most reliable investigation on diagnosing intraperitoneal abscess.

4. Perforation associated with haemorrhage

The association of a perforation with massive haemorrhage is grave but fortunately a rare complication. It may present on one of the three ways:

- Haemorrhage and perforation occurring concomitantly.
- Haemorrhage following a recently sutured perforation.
- Perforation occurring during the medical treatment of haemorrhage.

The clinical features are that of acute perforated peptic ulcer associated with signs of haemorrhage.

CLINICAL FEATURES OF DUODENAL ULCER PERFORATION[10,11,12,25]

Age: Duodenal perforation is rare before adolescence, common in 30-40 years age group.

Sex: More common in men than women.

History of Present illness :

- **Time of onset:** Very often the patient is able to exact the time of onset of perforation, common particularly after an exertion in the evening.
- **Mode of onset:** Sudden in onset, at times the patient may wake up from the sleep, due to onset of pain.
- **Pain:** Pain is tearing in the abdomen, intense in the epigastrium then spreads all over the abdomen.
- **Shifting of pain:** The pain shifts to right iliac fossa as the fluid flows along the right paracolic gutter to settle in right iliac fossa, thus mimicking appendicitis.
- **Radiation of pain:** Pain in peptic ulcer perforation is referred to the tip of the shoulder.
- **Nausea:** Present in some cases.

- **Vomiting:** Initially reflex vomiting occur due to irritation of nerves in the peritoneum and mesentery. In the later stages the vomiting is due to toxin action at the medullary centers and causing paralytic ileus. The vomiting then contains undigested food materials and occasionally blood when hemorrhage is present.
- **Bowels:** In the later stage, there may be desire to defecate due to irritation of retrovesical pouch by irritant fluid. Malaena occurs when the hemorrhage is associated with perforation.
- **Micturition:** Oliguria is present if patient is in shock.

Past History

In 80% of patients, there is a past history of dyspepsia of variable duration and in about 59% the perforation is recurrent. In the rest of the cases, the perforation may be the early clinical manifestation of a silent peptic ulcer.

Physical Examination

- **General Appearance:** In the initial shock of perforation, the face is pale livid with sweating.
- **Decubitus:** The patient lies in a characteristic posture of supine, rigid and immovable, refusing of any attempt to shift his postures.

- **Pulse:** Initially it is normal, rapid when peritonitis sets in and thready when the prognosis is grave.
- **Respiration:** Initially there is no change, becomes rapid and shallow when peritonitis sets in .
- **Temperature:** Initially normal, rises with the onset of peritonitis.
- **Tongue:** Usually moist, become dry and brown when the peritonitis sets in.

Examination of Abdomen

- **Respiratory movements:** Thoracic movement predominants over the abdominal movement with respiration.

- **Rigidity of abdomen:** Rigidity of abdomen is constant, continuous and characteristic. It is due to reflex contraction of the abdomen with predominance in the epigastrium and right hypochondrium. Rigidity is less in poor risk cases.

- **Liver dullness:** Obliteration of liver dullness elicited in front and in midaxillary line, is characteristic of this abdominal catastrophe in the second stage.

- **Free fluid:** Free fluid is present in variable degree on many acute abdominal conditions. When internal hemorrhage is excluded, fluid of appreciable amount points out the provisional diagnosis of perforation in acute abdomen.

- **Rectal examination:** There may be fullness in rectovesical or rectovaginal pouch

INVESTIGATIONS

The diagnosis is easy, but difficulties are experienced at times when the other conditions mimic the perforations are met with.

1. Plain x-ray

Plain x-ray of the abdomen in sitting and left lateral position confirms the diagnosis in 80% of cases by demonstrating free air under both domes of diaphragm. Krupasindu Panda and Chakrabothy (1976) reports subphrenic gas in 85% of cases.

According to Peter et al. (1955) and Isle (1951), X-Ray should be taken only after complete aspiration of food material from the stomach, as the mucous or food material may block the leak, hence results in absence of subphrenic gas shadow.

2. Gastroduodenogram

Some clinics have used X-ray pictures of abdomen following injection of 60ml of 50% gastrograffin through nasogastric tube. The dye escapes through perforation, thus enabling to demonstrate the site and size of perforation, evidence of chronicity, associated gastric ulcer and if any second ulcer present.

3. Ultrasound Examination[26]

Ultrasonography of abdomen was performed using a convex multi frequency probe (3.5-5mHz). Evidence of intraperitoneal free fluid and of reduced intestinal peristalsis was considered indirect evidence of perforation.

4. Computerized Tomographic Examination[26]

Because of classical presentation in most patients, CT-Scanning is rarely required for diagnosis. However, patients with perforated duodenal ulcer who are on steroid therapy or who are hospitalized for other abnormalities may develop occult causes of abdominal pain and sepsis. Gastrograffin swallow and/or CT-Scanning may be required to determine the cause on occult abdominal sepsis.

5. Serum Amylase

It is usually normal, but may be raised to small extent. Normal value of serum amylase is 80-180 somogyi units. Above 200units is considered pathological. Mortality is high for gastric and duodenal perforation with high serum amylase.

Helicobacter Pylori infection diagnosis[29,30]

The diagnosis of Helicobacter pylori infection is done by following different methods:

1.Non-invasive: a)Serology - ELISA.

b)Urea breath test.

2.Invasive: a)Rapid urease test e.g. Eco, Pyloritek

b)Histology

c)Culture.

Serology: H-Pylori infection evokes both local and systemic immune response. Serological tests can be done for detection of IgM, IgG, or IgA antibodies. The systemic IgG response is the most commonly used parameter for this infection.

ELISA, using a commercial kit, has high sensitivity (100%) and specificity (upto95%).

Urea breath test: This test is based on the production of urease by H- Pylori. The patient ingests a solution of urea containing a labelled carbon atom. The appearance of labelled carbon dioxide in the breath indicated the presence of infection. The label used is either non-radioactive ^{13}C or radioactive ^{14}C.

Rapid urease test: This test depends on the ability of H-Pylori to produce the enzyme urease, which hydrolyze urea to produce carbon dioxide and ammonium ions, which change the colour of the pH indicator phenolred from yellow to red indicating positive result.

Histology: H-Pylori can be identified on haematoxylin and eosin, modified Giemsa and Ethin-stony silver stains.

Culture: This is the most difficult method for diagnosing the H-Pylori infection. Successful growth of H-Pylori depends on laboratory expertise, timely handling of specimens, use of appropriate media and incubation environment. Culture facilitates are absent in most centers in India.

Abdominal Paracentesis

Diagnostic peritoneal tap is a simple procedure, which can be done quickly in cased of suspicious hollow viscous perforations. Four quadrant abdominal paracentesis has to be done.

DIFFERENTIAL DIAGNOSIS[11,12,13,16,17,30]

The duodenal ulcer perforation has to be differentiated from the following conditions, which can be divided into: Acute medical conditions and acute surgical conditions.

Acute Medical Conditions

1. Pleurisy:
2. Acute Pericarditis.
3. Gastric crisis or Tabes dorsalis.
4. Lobar pneumonia.
5. Acute alcoholism
6. Coronary thrombosis

Acute Surgical Conditions

1. Acute exacerbation of duodenal ulcer
2. Acute Pancreatitis.
3. Acute gastritis
4. Peritonitis from Acute appendicitis
5. Acute intestinal obstruction
6. Biliary colic in acute cholecystitis

7. **Raptured aortic aneurysm**

8. **Raptured ectopic gestation**

9. Perforated typhoid ulcer

TREATMENT

The perforation of duodenal ulcer is common problem, the most important and immediate step in the management is adequate resuscitation of patient on admission.

Two methods of treatment are practiced:

1. Non-surgical or conservative management,
2. Surgical management.

1. Open surgery

A. Simple closure of perforation.

B. Closure of perforation with definitive surgery.

- Truncal vagotomy with gastrojejunostomy.
- Antrectomy with vagotomy.
- Pyloroplasty with vagotomy.
- Partial gastrectomy with vagotomy.
- Highly selective vagotomy.

II. **Laparoscopic surgery**

Immediate management (Resuscitation)

1. If patient is in shock, elevate foot end of the bed, start intravenous fluids immediately with Dextran or Saline/Plasma/expanders.

2. A nasogastric tube is passed and the contents of stomach aspirated and Repeated Every Half An Hour

   To prevent further soiling of peritoneum

   To prevent aspiration of gastric content into lungs

   To decompress the stomach.

3. Bladder catheterization done in all patients to monitor urinary output.

4. Blood sample collected for grouping and cross matching, complete haemogram, blood urea, serum creatinine, and serum electrolyte study.

5. Blood pressure and pulse rate and urinary output should be recorded at an half hourly intervals.

6. Appropriate antibiotics (broad spectrum) should be given. Third generation cephalosporin and metranidazole are preferred.

7. Preparation of abdomen to be done

OPERATIVE MANAGEMENT[10,11,12,13,16,17,31]

Perforation of duodenal ulcer is usually treated by surgery.

Simple closure

After adequate resuscitation patient can be posted for surgery.

Anaesthesia: General anaesthesia, by IV thiopentone and Scolene, endotracheal intubation and maintenance by N_2 and O_2.

Procedure: Patient in supine position, abdomen opened with upper paramedian or upper midline incision. Bailey points out that in 10% of the cases, muffled pop of escaping gas can be heard on opening peritoneum. The free fluid is sucked and mopped with moist packs. The stomach is held near the greater curvature with a moist pack and search for perforation. The anterior part of the duodenum and distal stomach are inspected first, usually the perforation is found on the anterior surface of first part of duodenum. It is closed with interrupted suture using absorbable suture Material. After peritoneal toilet, two drains are placed, one in the right side near the perforation site and the other one in pelvis. The abdomen incision is then closed in layers.

Methods of closure of perforation

1. Simple closure:
    - Indicated for small ulcers with little induration and healthy tissue around. - Converts ulcer into a linear scar
    - More rapid healing.
    - Early remission of symptoms.
    - Absorbable / delayed absorbable /- Purse string suture to be avoided.
    - Inversion / eversion to be avoided.
    - Suture should be applied in the long axis of the gut to avoid narrowing of lumen.
    - Omentum is used for reinforcing the perforation.
2. Cellan Jones technique, Graham technique: Here free omental flap is used. Greater omentum is placed over the perforation and perforation is sealed by omentum.
3. Dragging the omentum into perforation and plugging it into the Ryles tube.
4. Use of rectus muscle to seal the perforation.

DEFINITIVE SURGERY

This has been advocated as it has been found that patients treated with simple closure have a severe relapse of the disease in >50% of cases in 5 years follow-up.

Indications for definitive surgery:

1. **Definite indications:**
    - Coexistent of perforation and obstruction.
    - Previous operation for perforated duodenal ulcer.
    - Perforated gastric ulcer with suspicion of malignancy.
    - Coexistence of haemorrhage and perforation.
    - Perforation of ulcer during medical treatment.
    - Combined gastric and duodenal ulcer, one of which has perforated.
2. **Relative indications:**
    - Young patient less than 45yrs.
    - Smoker.
    - Absence of purulent peritonitis.
    - If patient has reported before 8hrs after perforation.
    - Minimal peritoneal soiling.

Contraindications for definitive surgery:

- More than 24hrs of presentation.
- Poor risk patient.
- Concurrent medical illness

Advantages of definitive surgery:

- Reperforation is avoided.
- Second operation is avoided.

Gastric stasis after simple closure is avoided. - Postoperative pyloric stenosis of obstruction due to inflammation oedema is avoided. - In haemorrhage with ulcer perforation, - haemorrhage is cured.

Disadvantages of definitive surgery:

- It is more operative trauma to patient.
- It may be unnecessary in 10-15% of patient.

Types of definitive surgery

1. Truncal vagotomy with gastrojejunostomy.
2. Antrectomy with vagotomy.
3. Pyloroplasty with vagotomy.
4. Partial gastrectomy with vagotomy.
5. Closure with highly selective vagotomy.

1. Truncal vagotomy with gastrojejunostomy:

In this, after simple closure of perforation, peritoneum over the oesophagus is opened and left lobe of liver is mobilized detaching its coronary ligament and bilateral truncal vagotomy is done. Truncal vagotomy is always performed with a drainage procedure, because of rise of gastric stasis and delayed gastric emptying. The posterior surface of stomach is brought into infracolic compartment by opening the transverse mesocolon. Short loop of jejunum from duodenojejunal flexure is taken and approximated with the stomach and a classical GJ is performed. Rent in the mesocolon closed in layers.

2. Antrectomy with vagotomy:

Here, the distal half of the stomach is resected with Billroth II anastamosis.

3. Pyloroplasty with vagotomy:

Here, after excising perforation, Heineke, Mikulicz or Finney type of phloroplasty is performed and with vagotomy.

4. Partial gastrectomy with vagotomy:

When perforation is in lower gastric area, this procedure is done, followed with Billroth I anastomosis and vagotomy.

5. Highly selective vagotomy:

The objective of highly selective vagotomy is to denervate the parietal cell mass while preserving the vagal supply to the antrum, thereby avoiding drainage procedure. Preserving the anterior and posterior nerves of Laterjet does this, which provide the motor innervation to antrum. This procedure is done in case of perforation associated with haemorrhage.

In case of difficult closure of duodenal stump, the duodenal stoma can be placed either laterally or terminally. Tube duodenostomy using Kehrs "T" tube or end duodenostomy using Foleys catheter.

LAPROSCOPIC CLOSURE OF PERFORATION[27,28]

Recent development in minimal invasive surgery now allows laparoscopic approach to the patient with perforated duodenal ulcer.

The perforation can be approached using three additional ports. The perforation can be dealt by: - Fibrin glue for minute perforation.

Simple closure with omental patch and copious irrigation of the abdominal cavity.

Automatic staples suture can be applied via laparoscope.

A proximal gastric vagotomy or Taylor procedure (anterior seromyotomy and - truncal vagotomy) may be performed.

POSTOPERATIVE MANAGEMENT

- Nil by mouth till the intestinal sounds is regained.
- Nasogastric tube aspiration till it becomes less and bowel sounds - recover.
- Antibiotic covering anaerobic and aerobic organisms.
- Intravenous fluid therapy.
- H_2 blockers.
- Chest care.
- Drainage tube care.
- Watch for any evidence of intra-abdominal collection in case of postoperative fever.

POSTOPERATIVE COMPLICATIONS

- Pulmonary complications like atelectasis, pneumonia.
- Residual abscesses like subphrenic abscess, pelvic abscess.
- Peritonitis.
- Paralytic ileus.

- Early reperforation and leak, duodenal fistula.
- Deep vein thrombosis and pulmonary embolism.
- Renal failure.
- Mediastinitis.

Management of early perforation and leak

- Priority towards fluid and electrolyte management. - Nasogastric aspiration.
- H_2 blockers.
- Antibiotics.
- Nutrition of the patient, ideally total parental nutrition (TPN) or feeding jejunostomy.

Causes of leak are**:**

- Old patients.
- Large perforation.
- Inadequate closure.
- Difficulty closure with friable margin.
- Late presentation to hospital after perforation.

Leaks are usually seen from 2nd to 5th postoperative day presenting as bilious drain from the drain site.Fistula may be high or low

output. In case of high output fistula, where TPN facilities are available, patients can be managed conservatively, feeding jejunostomy can be added for enteral feeds. Trial can be given for 3weeks after which operative can be adopted.

Where TPN facilities are not available, ideally after resuscitation patient can be taken up to surgery at an early stage to prevent further deterioration. In low output fistula, conservative management is usually adopted.

Surgical management

- Feeding jejunostomy.
- Use of serosal patch (Kobold & Thal) technique: The upper jejunum is serosa used as a loop or Roux en y loop to occlude the perforation.
- Partial gastrectomy: In large perforation with friable margins where repair cannot be done, it is ideal to proceed with partial gastrectomy with polya anastomosis.
- Conservative or non-operative management: By passing Foley's catheter into the drain wound and manipulating it into or near to the perforation can be converted into a controlled fistula.

H2 Blocker antagonist: These drugs act by selectively blocking H2 receptors of parietal cells. They have a dose dependent antisecretory potency. Their simple dosage schedules and associated good therapeutic compliance.

Drugs: Cimetidine: No more used.

Ranitidine: 150mg Bid, after 4-6wks OD at night.

Famotidine: 20-40mg OD.

Roxatidine: 75mg OD.

Nizatidine: 20mg Bd.

Proton pump inhibitors: These act by inhibiting the H+ / K+ ATP system on the luminal side of the parietal cells. Action is long lasting and dose dependent. In dose of 20-40mg OD, it achieves almost 100% inhibition of intragastric acidity throughout day and night.

Dosage: 20-40mg OD for 4-6weeks,followed by 10-20mg OD.

H-PYLORI INFECTION ERADICATION[13,16,32,33,34,35]

The key success factor in management of peptic ulcer is treatment of H-Pylori infection, which has been widely advocated. Current regimens for eradication of H-Pylori infection are quite diverse, not only in the combination of agents used but also in dosage and duration of the treatment.

There are various regimens against H-Pylori:

1. Dual drug therapy.
2. Triple drug therapy.
3. Quadruple drug therapy.

1. Dual drug therapy

• Proton pump inhibitor + clarithromycin/ amoxycillin.

• Ranitidine + clarithromycin for 14 days. Not recommended .

2. Triple drug therapy

- Omeprazole 40mg OD + Clarithromycin 500mg BID +Metranidazole 400mg BID for7days.

- Omeprazole 40mg OD + Amoxicillin 500mgBID +Clarithromycin 500mg BID for 7 day .

- Omeprazole 40mg OD + Amoxicillin500mg BID +Metranidazole 400mg BID for7–10days.

- Colloidal Bismuth Subcitrate 125mg QID for 14 days +Amoxicillin500mg BID + metranidazole 400mg BID

3. Quadruple drug therapy

Omeprazole 40mg OD + Collidal Bismuth Subcitrate 125mg QID 4OD +Tetracycline 500mg TID + Metranidazole 400mg TID for 7days.

The Clarithromycin based regimens are much costlier than Amoxicillin based regimens.

NON-OPERATIVE OR CONSERVATIVE MANAGEMENT[36]

In majority of patients surgery remains treatment of choice. In certain situations, conservative management should be considered.

Indications for conservative management:

1. Where general condition of the patient is bad, risk of general anaesthesia is considered too great.
2. Lack of surgical facilities.
3. Clinical signs suggesting only of minimal spillage with sealed perforation which has been shown by gastrograffin radiograph.

Contraindications for conservative management:

1. Perforation in presence of steroids, which would diminish the patients ability to heal the ulcer spontaneously.
2. Gastric ulcer.
3. Patients who have continued leakage on gastrograffin radiograph.
4. Patients who perforate while on active antacid therapy.
5. Uncertain diagnosis.

Conservative management consists of:

- Nil by mouth
- Continued nasogastric aspiration
- Intravenous fluids
- Intravenous H2 receptor antagonist
- Appropriate antibiotics
- Appropriate sedative.

If the distension of abdomen increases with the condition of deteriorating, then the flank drain (bilateral) may be placed under local anesthesia, to drain the fluid.

Advantages of conservative management:

- Operation can be avoided.

- A percentage of patients do not need any further definitive operation, in such patients, unnecessary operation can be avoided.
- In few patients, when perforation will get sealed off and such patients would be benefited.

Disadvantages of conservative management:

- The site of perforation usually remains in doubt.
- The nature of underlying condition (benign or malignant) remains uncertain.
- Recurrence of ulcer symptoms (Illingworth, 1994)
- Recurrence of perforation.
- Risk of detoriation.

PROGNOSIS[37-44]

Many factors influence the speed at which the peritoneal crisis develops.

Mortality depends upon the following factors:

Age: Mortality increases with increasing age (Illingworth, 1994).

General condition of patient: Poor general condition of the patient carries high mortality.

Presentation with shock: (Systolic BP <90 mmHg) Patients presenting with shock have a high mortality rate.

Boey John et al revealed concurrent medical illness, preoperative shock and delayed operation (>48hours) as significant risk factors that increase mortality in patients with perforated duodenal ulcers (1982).

Presentation with renal failure: May be hypovolaemic/septicemic origin. Oliguria carries high mortality rate.

Ulcer history: Patient with long ulcer history have shown to carry high risk and concomitant bleeding and perforation also carried high mortality rate.

Concomitant medical illness: Concomitant cardiac, pulmonary, renal and other diseases in association with perforation carry high mortality rate.

Duration of perforation: If the duration is longer, higher is the mortality.

In the presence of gross contamination, late exploration (after 48hours) carried a high mortality i.e. 50% (Arthue A. Cicola et al, 1999). The importance of the peritoneal soilage and duration of perforation is mentioned as a risk in the outcome of the perforation of duodenal ulcer (Donaldson, 2000). Bharati C Ramesh et al reported that 12% of patients reached the hospital within 12 hours, 40% reached hospital within 25-48 hours and 24% after 48 hours (IJS, 2006). Brazynski M et al reported that 48.15% patients to hospital after 24 hours of perforation (1999).

Size of perforation: In duodenal ulcer, the size varies from 3mm to 1cm in diameter. Larger the perforation and older the patient, the higher the morbidity and mortality.

Number of perforation: Perforation is always single, but there are reports of more than one perforation. More than one perforation, then higher the morbidity and mortality.

Nature of gastric contents, kinds of microorganism which predominant:

(Bacteriology)[45-48]

The peritonitis resulting from perforation of peptic ulcer is at first often non-infectious and is due to instant action of the gastric and duodenal ulcer contents and thus the infective peritonitis in early case is unlikely. However, it depends on the general condition of the patient and the resistance to infection. Grarco and Chawo in 1974 and Boey and colleagues in 1982 found that more than half the cultures of peritoneal fluid taken at the time of operation were sterile. The preoperative use of antibiotics may play a considerable part in these findings. It appears bacterial peritonitis is seen only in grossly neglected cases.

Postoperative complications

a) Pulmonary complications like bronchopneumonia, atelectasis and pulmonary embolism are the important and frequent complications associated with high mortality and morbidity.

b) Wound complications and intra-abdominal collections also increase the morbidity.

MATERIALS AND METHODS

Between August 2013 and December2013, all patients admitted in Rajiv Gandhi Government General Hospital and diagnosed with acute perforated peptic ulcers were recruited into a prospective study.Informed and written consent was obtained before the study. Demographic data, medical history, past history of previous peptic ulcers, and the use of non-steroidal anti-inflammatory drugs (NSAIDs) were recorded. All patients after resuscitation were treated by laparotomy and a simple closure of the perforated ulcer by oversewing with greater omentum. Biopsy taken from the ulcer site edge and adjoining the mucosa. The biopsy material was preserved in 20% formalin and the specimen was sent as early as possible for histopathological examination by Giemsa staining. Extensive cleaning of the peritoneal cavity with several litres of warm sterile saline solution was performed and simple omental patch closure done

Patient data collection and evaluation :

- Detailed history of patient will be entered in proforma.
- Complete haemogram, LFT, prothrombin time, Sr.electrolytes, Sr.Amylase serology, blood urea, serum creatinine will be sent.

- Preliminary, X-ray, abdomen (erect) and lateral decubitus for patients who Cannot stand, will be done on the same day of presentation..
- Patient will be informed about any surgical procedure and consent will be taken.
- Patient will be operated after adequate resuscitation.
- Ultrasound / CT abdomen and pelvis(if necessary) will be done immediately .
- Once the clinical and imaging evidence shows signs of perforation patient will be taken up immediately for surgery(Laporotomy and Gastro duodenal ulcer edge / mucosal biopsy).

Inclusion criteria

1. All Patient of Gastro duodenal perforation diagnosed clinically,radiologically, followed by explorative laparotomy for repair.

Exclusion criteria

1. Patient with H/o trauma are excluded.

Sample size: 50 cases

Fig.11 : Picture showing distended abdomen with board like rigidity

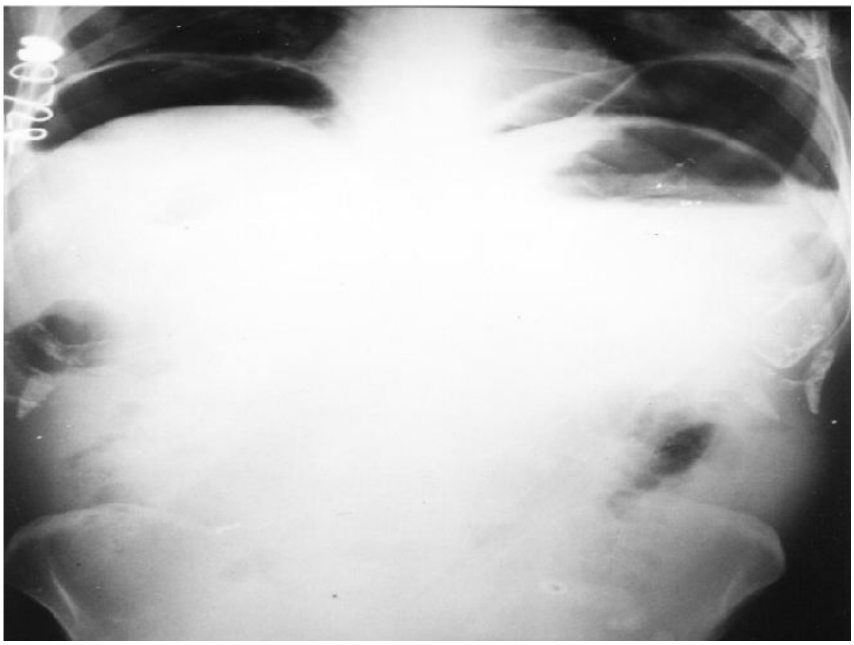

Fig.12 : Picture showing erect abdomen x-ray with gas under the diaphragm

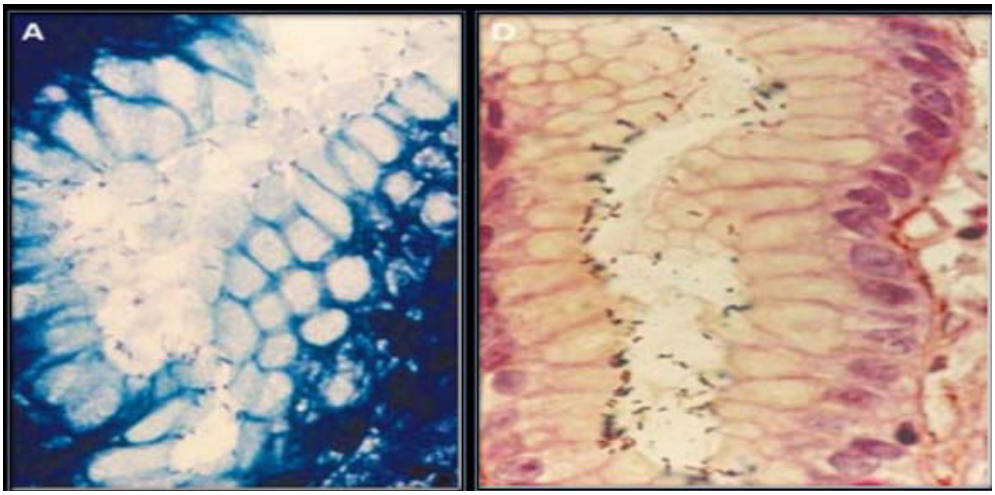

Fig.13 : Demonstartion of H.pylori by staining technique in perforated ulcer

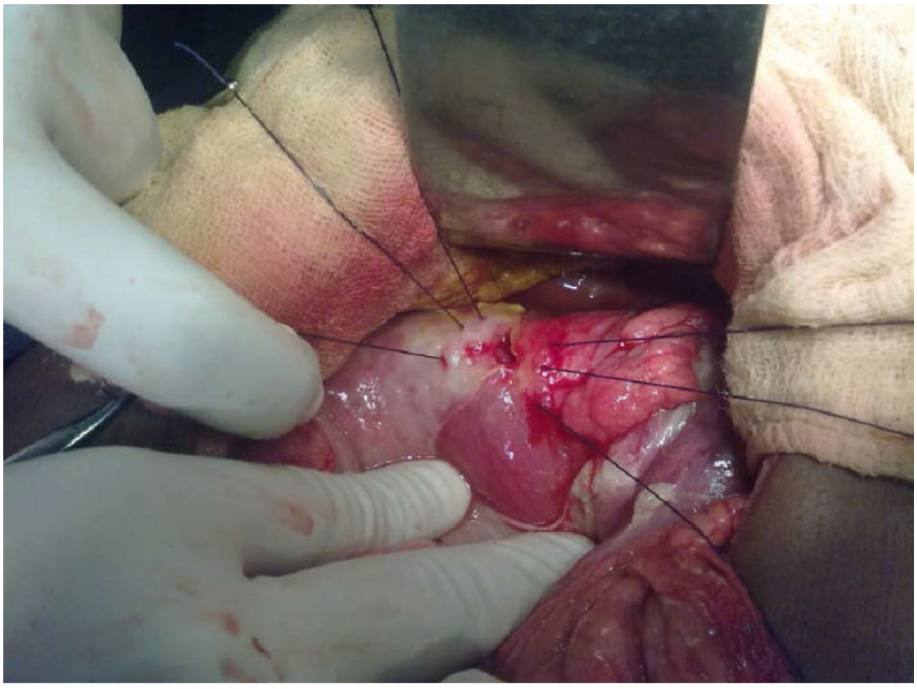

Fig.14 : Simple closure in duodenal ulcer perforation

OBSERVATION

The Gastroduodenal ulcer perforations are one of the common surgical emergencies.

From August 2013 to December 2013, a total of 50 patients with gastro---duodenal perforation were studied from surgical units of **RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL.**

Age : The age incidence of 50 patients's analysis is as follows :

Table-1 : Age Distribution of Gastroduodenal perforation

Age in yrs	Perforation		%
	Duodenal N(%)	Gastric N(%)	
21-30	11(22%)	1(2%)	12(24%)
31-40	7(14%)	1(2%)	8(16%)
41-50	8(16%)	2(4%)	10(20%)
51-60	9(18%)	1(2%)	10(20%)
61-70	5(10%)	0	5(10%)
71-80	4(8%)	1(2%)	5(10%)
TOTAL	**44(88%)**	**6(12%)**	**n=50(100%)**

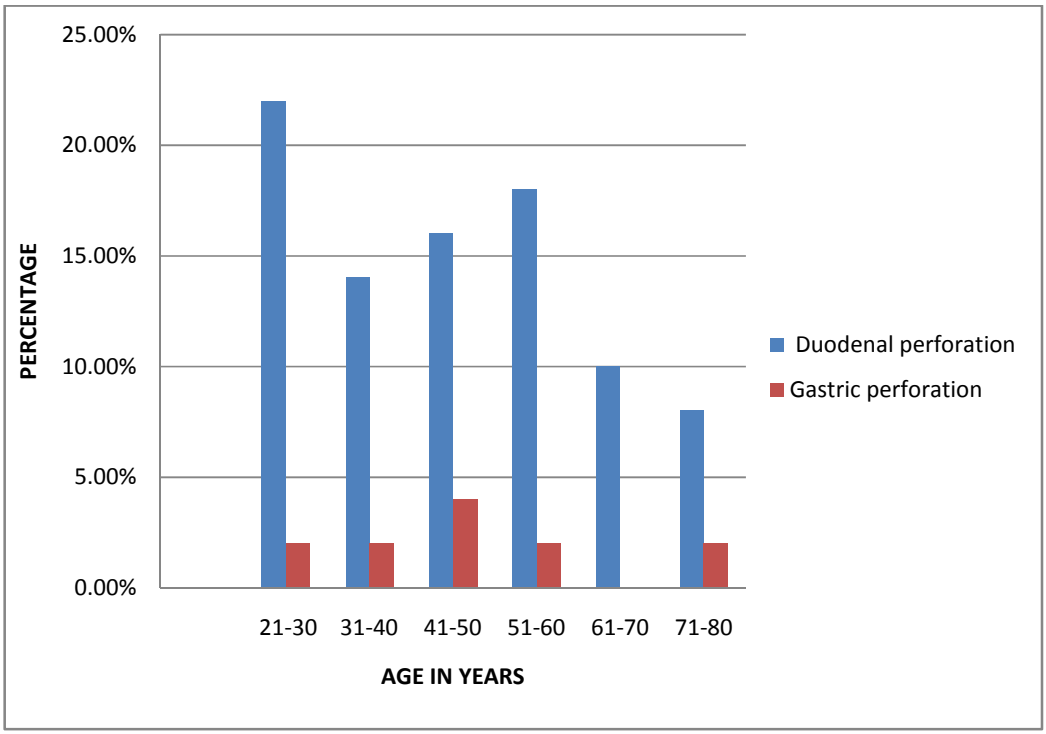

Fig.15 : Bar graph showing age distribution of Gastroduodenal perforation

In my study, highest incidence of gastroduodenal perforation is seen in 3rd decade of life. This maybe due to the stress and strain during the same period.

Table-2 : Age Distribution of H.Pylori in Gastroduodenal perforation

Age in yrs	H.Pylori biopsy		%
	Positive N(%)	**Negative N(%)**	
21-30	3(6%)	9(18%)	12(24%)
31-40	2(4%)	6(12%)	8(16%)
41-50	6(12%)	4(8%)	10(20%)
51-60	6(12%)	4(8%)	10(20%)
61-70	1(2%)	4(8%)	5(10%)
71-80	4(8%)	1(2%)	5(10%)
TOTAL	**28(56%)**	**22(44%)**	**n=50(100%)**

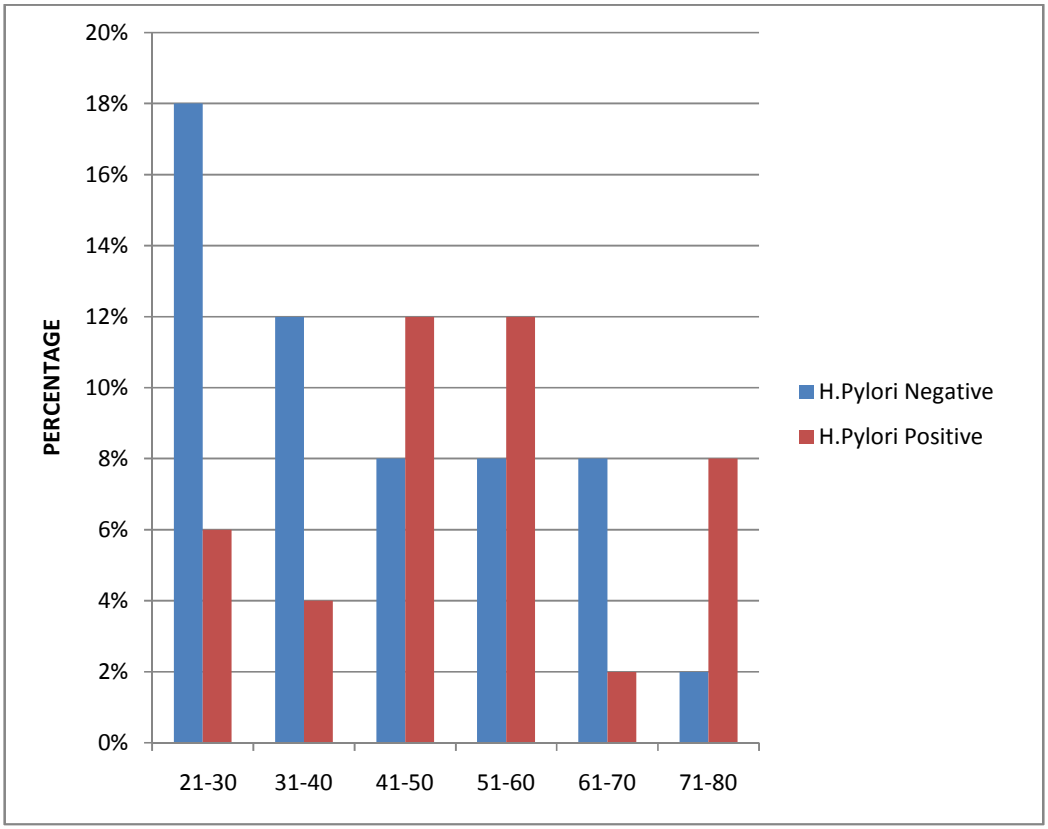

Fig.16 : Bar graph showing Age Distribution of H.Pylori in Gastroduodenal perforation

In my study, H.Pylori infection has maximum incidence in 2nd and 3th decade of life in patient presenting with Gastroduodenal perforation.

Table -3: Gender distribution of Gastroduodenal perforation

Gender	Perforation		Total %
	Duodenal	Gastric	
Female	2(4%)	0	2(4%)
Male	42(84%)	6(12%)	48(96%)
Total	**44(88%)**	**6(12%)**	**n=50(100%)**

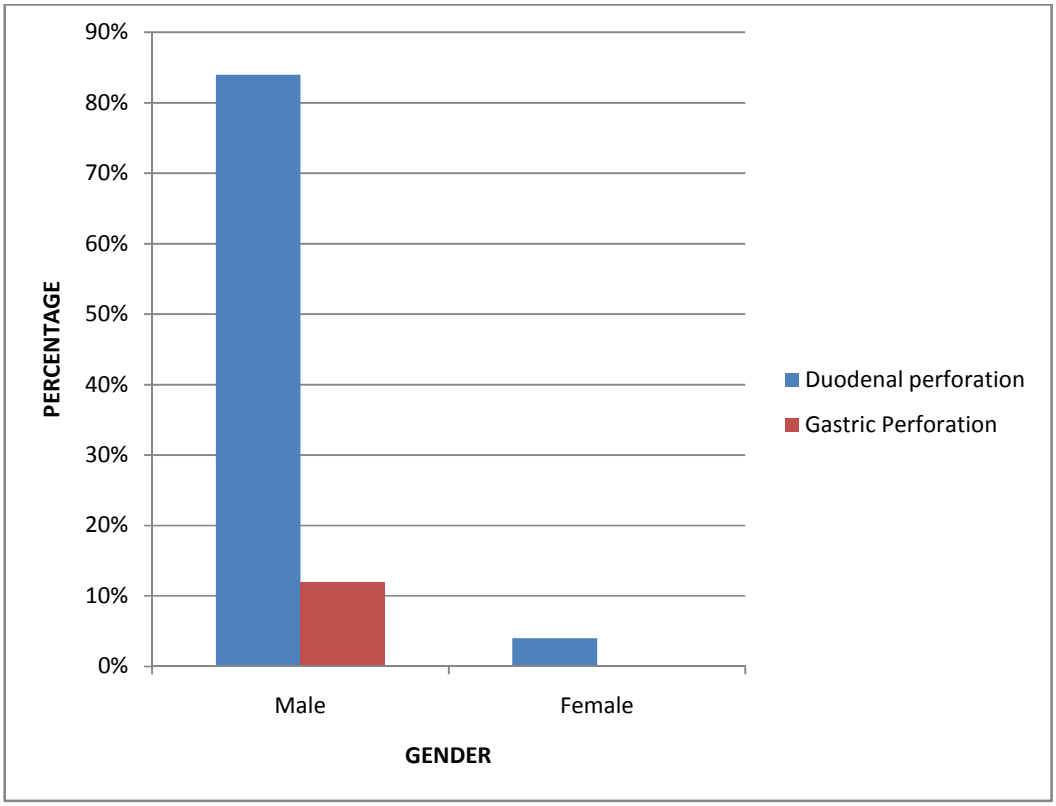

Fig .17 :Bar graph showing Gender distribution of Gastroduodenal perforation

In my study, Gastroduodenal ulcer perforation is more common in males i.e., Duodenal perforation – 42(84%) & Gastric perforation – 6(12%)

Table-4 : Gender distribution of H.Pylori in Gastroduodenal perforation

Gender	H.Pylori biopsy		Total
	Negative	Positive	
Female	2(4%)	0	2(4%)
Male	26(52%)	22(44%)	48(96%)
Total	**28(56%)**	**22(44%)**	**n=50(100%)**

Table-5 : Risk factors

RISK FACTORS	PERFORATION		Total %
	Duodenum	Gastric	
Smoking	42(84%)	6(12%)	96%
Alcohol	42(84%)	6(12%)	96%
NSAIDs	9(18%)	0	18%

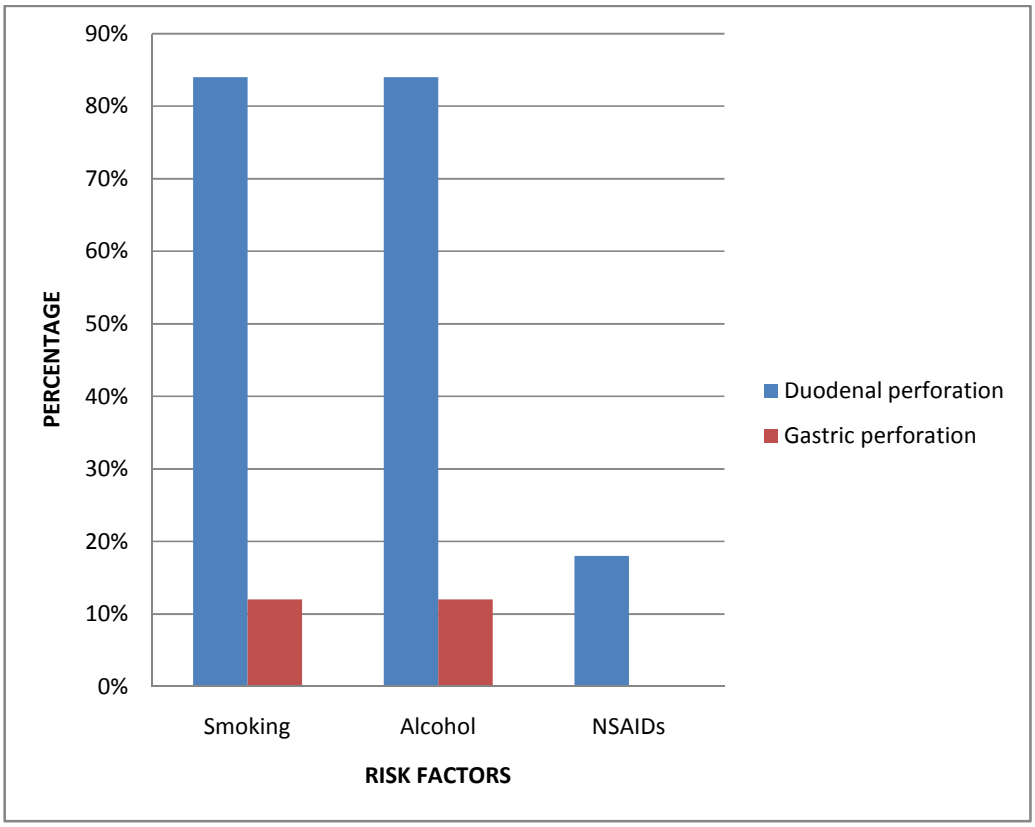

Fig.19 : Bar graph showing riskfactors

Among 50 patients 48 patients had history of smoking and alcoholism.

The incidence is more in case of smokers and alcoholics.

Table -6 : Mode of Presentation : Symptoms

Symptoms	PERFORATION		Total %
	Duodenum	Gastric	
Abdominal pain	44(88%)	6(12%)	100%
Abdominal distension	44(88%)	6(12%)	100%
Vomiting	36(72%)	4(8%)	80%
Fever	28(56%)	4(8%)	64%

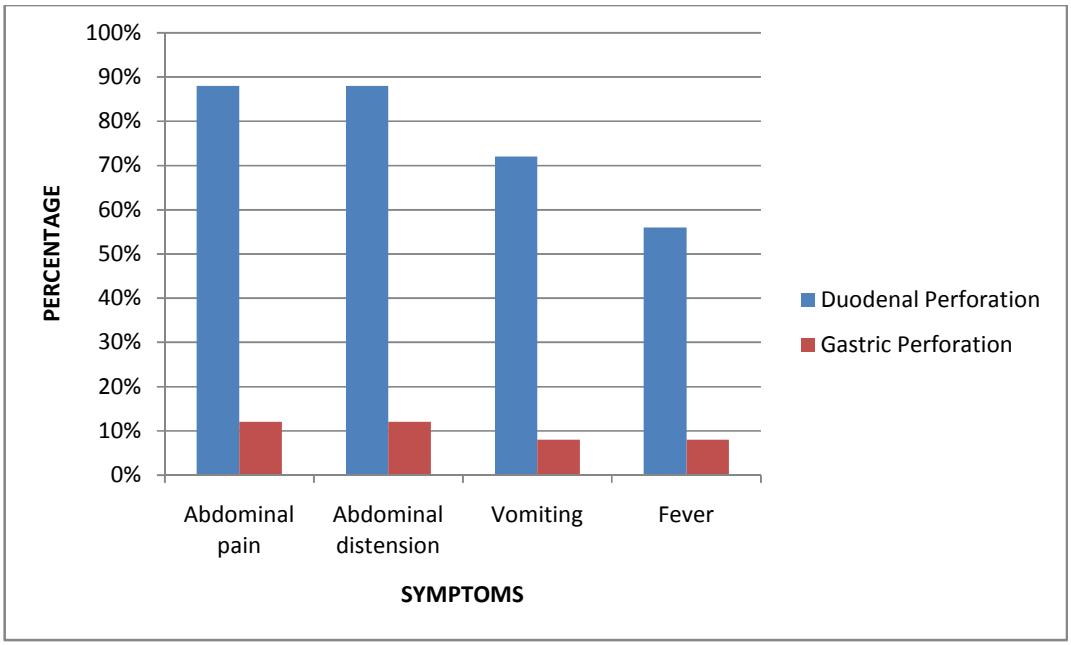

Fig.20: Bar graph showing Mode of presentation - Symptoms

Table-7 : Mode of presentation - Signs

Signs	PERFORATION		Total %
	Duodenum	Gastric	
Guarding	44(88%)	6(12%)	100%
Rigidity	44(88%)	6(12%)	100%
Tenderness	44(88%)	6(12%)	100%
Obliteration of Liver dullness	44(88%)	6(12%)	100%
Bowel sounds absent	44(88%)	6(12%)	100%
Shock	38(76%)	6(12%)	88%

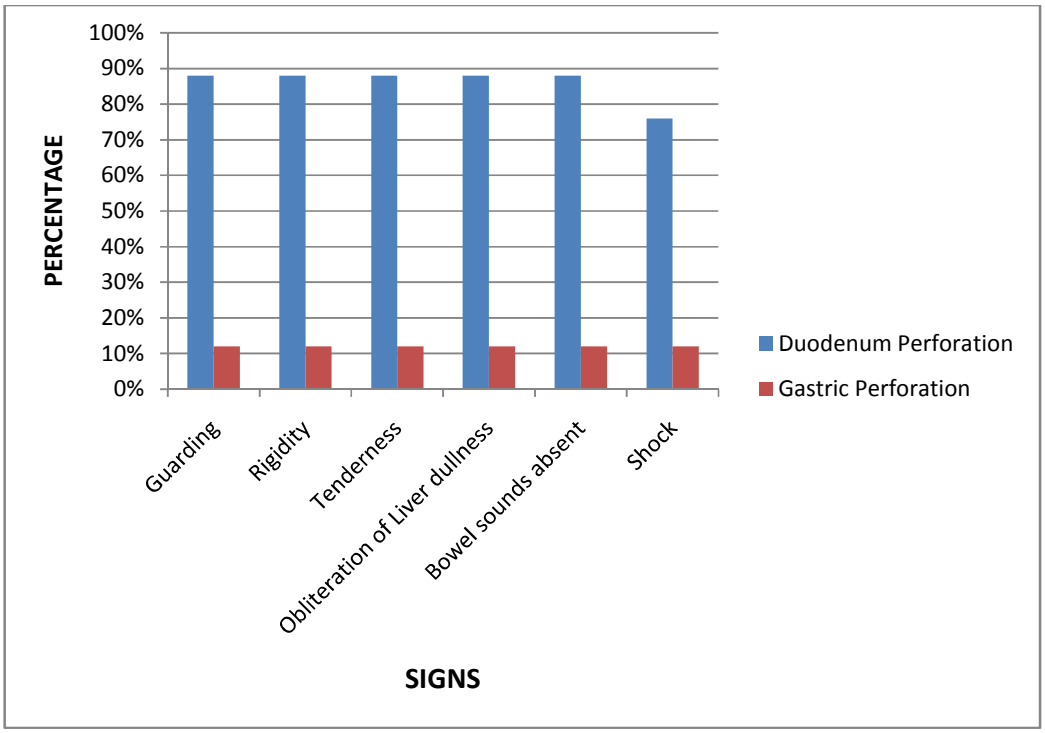

Fig.21: Bar graph showing Mode of presentation - Signs

Table-8 : H.Pylori prevalence in Gastroduodenal perforation

H.Pylori biopsy	PERFORATION		Total %
	Duodenum	Gastric	
Positive	21(42%)	1(2%)	44%
Negative	23(46%)	5(10%)	56%
Total	44(88%)	6(12%)	100%

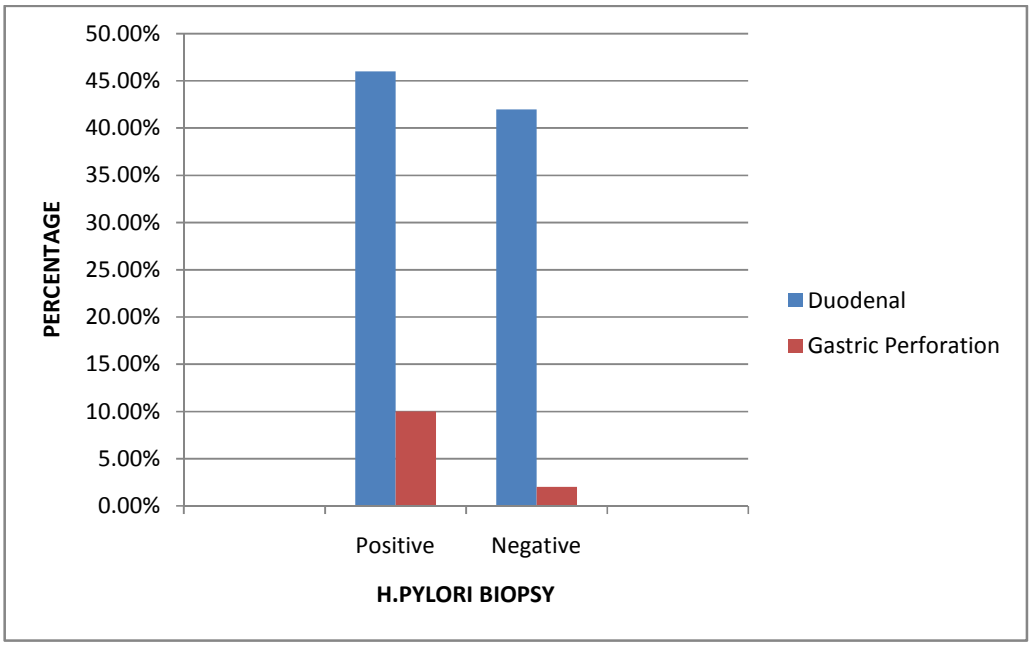

Fig.22:Bar graph showing Prevalence of H.Pylori in Gastroduodenal perforation

DISCUSSION

Gastroduodenal ulcer perforation is one of the commonest surgical emergencies.Although incidence of surgery for duodenal ulcer diseases has reduced drastically with advent of omeprazole and H2 receptor antagonist, but surgery for perforation has not changed.

Age incidence

Duodenal ulceration is common in the age group of 20 -30 years and 40-50 years in our study, but age is no bar for perforation to occur. It has also been reported in 4years old infant (Bhatacharya, 1969).[6]

Table -9 : Showing peak age incidence by various authors

Authors	**Peak age in years**
Palanivelu et al (2007)	20 –30
Philipo.l.Chalya et al (2011)	40 – 50
Present series(2013)	20 –30

Present series matches with Palanivelu et al[49] and Chalya et al.[50].

Savnes C[51] has reported that the lethality is higher in the elderly reported that age of a patient, rather than the type of surgery which

influences the mortality in a perforated duodenal ulcer and he reported the mortality rate of 0.6% in < 50 years age group, 15% in 50-60 years age group and 45.2% in > 60 years age group (2009).

Sex incidence

In our studied series 96% were males and 4% were females, and the male-female ratio being 24:1. Perforation is more common in males than females, because males were subjected to more stress and strain of life and female sex hormone offer some security against perforation as claimed by Skovgaard (1997).[52]. High prevalence of perforation is more is in male society as they are more in stress as compared to there female counterparts said by Zahid Amman (2008).[53] Our study matches also with Ng et al (1996)[54] and Aman et l (2002).[55]

Table-10 : Showing sex incidence by various authors

	Male : female ratio	
Primose N. Jhon (Baily and Love)	2	: 1
Palanivelu et al (2007)	12.3	: 1
Chalaya et al (2011)	1.3 :	1
Present series(2013)	20	: 1

Habits

Hermansson M and Ekedhal A et al[56] showed that smoking increased the risk of ulcer perforation to 10-fold in the age group of 15-74 years, and there was highly significant dose-response relationship. The results were similar in men and women and for duodenal ulcer perforation. Savens et al[51] concluded that smoking is a casual factor for ulcer perforation and accounts for a major part of ulcer perforations in the population aged > 75 years. Smoking is a definite risk factor for peptic ulcer perforation. In this study it showed that smoking has as a definite role in Duodenal ulcer perforation.

Table : Incidence of causal factors

Authors		Smoking	Alcohol	NSAIDS
Palanivelu et al (2007)		72.5 %	70.2 %	10 %
Chalya et al (2011)		64.3 %	85.7 %	10.7 %
Present series (2013)	DUODENAL PERFORATION	88 %	88%	18 %
	GASTRIC PERFORATION	12%	12%	0%
	TOTAL	100%	100%	18%

Clinical presentation of perforation

In the present study, pain was present in all cases of duodenal ulcer perforation, indicating that the pain was the most common symptom in duodenal ulcer cases. Guarding and Rigidity was present in 100% of cases. Which was comparable with the literature. All patients had absent bowel sounds.

Table : Clinical presentation of duodenal ulcer perforation by various authors

Clinical presentation	**Present series(2013)**			**Chalya et al (2011)**
	Duodenal Perforation	Gastric Perforation	Total	
Pain	88%	12%	100%	97.6%
Distension	88%	12%	100%	76.2%
Vomiting	72 %	8%	80%	36.9%
Tenderness	88%	12%	100%	88.1 %
Fever	56%	8%	64%	21.4 %

Prevalence of H-Pylori

Enders K & Lam et al (2000)[58] told 81% cases of perforated duodenal ulcer were infected with H.pylori. Yekin et al[59] (2010) noted that 80.9% of the patients with perforated duodenal were infected with H-pylori. Kate V et al[60] (2001, BJS) reported the 73% prevalence of H-pylori in perforated duodenal ulcer. In the present study, we were able to analyze the H-pylori infection by taking ulcer edge biopsy (histopathological examination), The incidence of H.pylori in present study was 44% Presence of H.pylori is Valuable in different study groups

Table : Incidence of H.pylori by various authors

Authors		Incidence of H.pylori
Lahm et al (2000)		81%
Palanivelu et al (2007)		60%
Chalya et al (2011)		64%
Present series (2013)	Duodenal Perforation	42%
	Gastric Perforation	2%
	Total	44%

CONCLUSION

- Gastroduodenal ulcer perforation still remains one of the most common cause of acute abdominal catastrophe, with male preponderance.

- We conclude that H. pylori is an important factor in the etiology of gastroduodenal ulcer perforation and accounts for 44 % of the cases. (Duodenal perforation – 42%, Gasstric perforation – 2%)

- In our study, we found that gastroduodenal ulcer perforation is a multifactorial disease with alcohol (64%), smoking (48%) and NSAIDs (22%) being common causative factors.

- Hence we conclude that , H.Pylori is one of the causative factor for the Gastroduodenal perforation.

SUMMARY

In this series, 50 cases of duodenal ulcer perforation cases were studied during the period from August 2013 to December 2013 at Rajiv Gandhi Government General Hospital, Chennai, admitted in all units in the Department of General Surgery.

Duodenal ulcer perforation commonly occurs in the age group of 20-30 years . Gastric ulcer perforation commonly occurs in 40-50 years.

Gastroduodenal ulcer perforation was common in males than females. 84% were males and 4% were females in duodenal ulcer. 12% were males in gastric ulcer perforation.

Smoking and alcohol beverage consumption were risk factors in most cases (96% and 96%) in causation of perforation of gastroduodenal ulcer, but NSAIDs is an emerging factor seen in 18% cases.

H.pylori study was positive 21(42%) cases of duodenal ulcer perforation, and 1(2%) case in gastric ulcer perforation. Hence it may play a role in gastroduodenal ulcer in perforation.

Alcohol and smoking are synergistic with h.pylori in duodenal ulcer perforation, 48 patients were smokers (96%) and 48 were alcoholics (96%).

Sudden onset of abdominal pain, situated at epigastrium and right hypochondrium was constant symptom. Vomiting and nausea were not uncommon.

Tenderness, rigidity, obliteration of liver dullness are the important signs. Absence of bowel sounds is one of the sign of perforative peritonitis.

Absence of previous history of peptic ulcer should not be considered as an important criteria to rule out the possibility of duodenal ulcer perforation, as sizeable number of patients do not give positive history of chronic duodenal ulcer.

Presence of gas under the diaphragm confirms the diagnosis, but their absence does not exclude the diagnosis.

Resuscitation and preoperative management of the patient is as important as the surgical procedure. The surgical management of duodenal ulcer perforation was mainly by simple closure of perforation with omental patch.

Risk factors for operation of perforated duodenal ulcer was old age, duration of perforation, size of perforation and presence of preoperative shock.

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S.No.	name	age	gender	ip no	Hb	tlc	dc	rbs	urea	reatinin	smoker	alcoho	NSAID	**lori bi**	x-ray	dominal p	minal dist	vomiting	guarding	rigidity	fever	owelsoun	ration of	endernes	shock	**of perfora**
1	machandra	54	m	99742	8.6	12000	79/20	112	45	1.3	Y	Y	N	**n**	p	p	p	a	y	y	a	a	p	p	a	**gastric**
2	boopalan	40	m	37931	8.6	6800	76/24	94	38	0.8	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
3	sivakumar	25	m	89961	8.6	7000	84/20	94	42	0.9	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
4	elumalai	29	m	39483	9.4	8600	80/20	92	37	0.8	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	p	**duodenum**
5	siva	40	m	61430	8.8	8700	79/20	86	41	0.9	Y	Y	Y	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
6	sriram	45	m	80937	8.6	9400	89/20	76	43	1.2	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
7	kumar	54	m	93571	9.4	9400	82/20	94	38	1.2	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
8	palpandi	40	m	10088	8.8	8700	93/7	88	42	1.3	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
9	sethu	27	m	78630	8.6	9600	86/24	94	37	1.1	Y	Y	Y	**n**	p	p	p	a	y	y	a	a	p	p	p	**duodenum**
10	saminathar	60	m	77312	9.3	8400	87/24	78	44	0.9	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
11	ithima bee	70	f	100588	8.6	7800	84/21	94	44	1.1	N	N	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
12	saravanan	25	m	85976	9.4	9600	86/4	86	41	1.2	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	p	**duodenum**
13	prakash	26	m	88957	8.9	7600	94/6	92	38	1.3	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
14	padmavath	70	f	49612	8	8900	72/17	116	41	1.1	N	N	N	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
15	murugan	35	m	28690	7.4	9600	86/20	96	44	1.2	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
16	smith	70	m	49612	9.2	7800	78/20	82	37	1.2	Y	Y	Y	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
17	anand	30	m	98637	8.2	8400	82/8	76	36	1.3	Y	Y	Y	**n**	p	p	p	a	y	y	p	a	p	p	a	**duodenum**
18	amodhara	36	m	74176	8.2	7800	78/20	89	42	1.2	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	p	**duodenum**
19	annamalai	38	m	37131	9.4	8200	86/14	84	39	1.1	Y	Y	N	**n**	p	p	p	a	y	y	a	a	p	p	a	**gastric**
20	arun kuma	26	m	47128	9	8600	86/14	78	41	0.8	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
21	anganatha	73	m	37950	10	8900	84/16	112	44	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
22	fakrudeen	27	m	89806	9.4	8700	86/14	94	41	0.9	Y	Y	N	**p**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
23	naveen	26	m	101593	8.6	7400	82/18	92	38	1.2	Y	Y	Y	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
24	rakshil	25	m	103860	8.8	7200	86l14	86	40	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
25	arokiyaraj	33	m	10621	9	9200	76/20	92	38	1.3	Y	Y	N	**p**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
26	jagan	80	m	39679	8.2	8000	79/20	112	41	0.8	Y	Y	Y	**p**	p	p	p	a	y	y	p	a	p	p	a	**duodenum**
27	anandan	50	m	32113	9	7800	76/24	94	36	0.9	Y	Y	Y	**p**	p	p	p	a	y	y	a	a	p	p	a	**duodenum**
28	raja	30	m	77557	8.4	7400	84/20	94	43	0.8	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**gastric**
29	safiq	34	m	83832	9	7600	80/20	92	40	0.9	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
30	rajan	73	m	88559	8	7400	79/20	86	38	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
31	kesavan	73	m	80567	8.2	7600	89/20	76	41	1.2	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**gastric**
32	ganesh	58	m	87248	8.4	7400	82/20	94	39	1.3	Y	Y	N	**p**	p	p	p	a	y	y	a	a	p	p	a	**duodenum**
33	raja sekar	62	m	87214	8	9200	93/7	88	38	1.1	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	p	**duodenum**
34	mani	55	m	92577	9	7800	86/24	94	47	0.9	Y	Y	Y	**p**	p	p	p	a	y	y	p	a	p	p	a	**duodenum**
35	munusamy	50	m	82044	8	7400	87/24	78	41	1.1	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
36	durai	53	m	90127	8.3	7200	84/21	94	42	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
37	joseph	46	m	96514	9.2	7200	86/4	86	39	1.3	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**gastric**
38	selvam	60	m	67847	9	7600	94/6	92	42	1.1	Y	Y	N	**p**	p	p	p	a	y	y	a	a	p	p	a	**duodenum**
39	iyyappan	42	m	72087	9	9200	72/17	116	41	1.2	Y	Y	N	**p**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
40	ari prasath	74	m	57906	9.2	7800	86/20	96	42	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
41	arumugam	54	m	67912	8.4	7600	78/20	82	62	1.3	Y	Y	N	**p**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
42	ndaramoor	48	m	78611	8.4	8600	82/8	76	44	1.2	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	p	**duodenum**
43	arasu	58	m	79191	9.6	8400	78/20	89	41	1.1	Y	Y	N	**p**	p	p	p	a	y	y	p	a	p	p	a	**duodenum**
44	rajendiran	50	m	81218	9.7	8200	86/14	84	42	0.8	Y	Y	N	**p**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
45	perumal	47	m	88719	9	7800	86/14	78	39	1.2	Y	Y	Y	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
46	thirupathi	47	m	88742	7.2	8600	84/16	112	38	0.9	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**gastric**
47	hussain	60	m	82174	9.1	7800	86/14	94	41	1.2	Y	Y	N	**n**	p	p	p	p	y	y	p	a	p	p	a	**duodenum**
48	arayanasar	65	m	62251	9	11900	82/18	92	39	1.2	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
49	marimuthu	46	m	72312	9	11400	86l14	86	29	1.1	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**
50	nanikandar	26	m	73129	9.4	9200	76/20	92	41	1.4	Y	Y	N	**n**	p	p	p	p	y	y	a	a	p	p	a	**duodenum**